THE
SURGERY OF THE STOMACH

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THE STOMACH

A HANDBOOK OF DIAGNOSIS
AND TREATMENT

BY

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LONDON

NEW AND REVISED EDITION

WITH PLATES

TO

MY FRIEND

Dr. WILLIAM J. MAYO

OF ROCHESTER, U.S.A.

TO
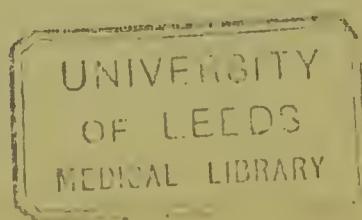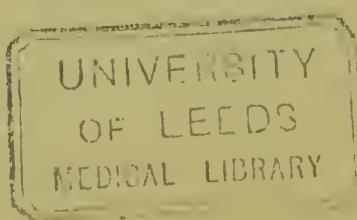
MY FRIEND

Dr. WILLIAM J. MAYO

OF ROCHESTER, U.S.A.

PREFACE TO THE SECOND EDITION

ALTHOUGH the first edition has been out of print for some months, the time which has elapsed since the original publication is so short that extensive revision is not called for. I have, however, endeavoured to profit by the criticisms of my Reviewers.

The photographs illustrating the operation of gastrojejunostomy have been reproduced in pairs for use stereoscopically, as was intended originally. The reader is urged strongly to look at them through a stereoscope,[1] as when used in this way every detail is clearly visible.

The chapter on the physiology of gastro-jejunostomy has been re-written in the light of further investigations on this subject.

Thanks to improvements in technique on the part of various workers, the value of radiographs in the diagnosis of abdominal lesions has been increased enormously. Accordingly several plates illustrative of the use of X-rays in diagnosis have been added, for which I have to thank my friend, Dr. A. C. Jordan, whose admirable work in this department of medicine is known so widely, both here and in America.

[1] May be obtained from Messrs. Aitchison & Co., 6 Poultry, London, E.C. 2*s*. 6*d*. each, post free.

Other minor alterations in, and additions to, the various chapters have been made, so that I hope the book may deserve in some degree the kind words which have been written concerning it.

Owing to my absence in America, the task of correcting the proofs and making a new index has fallen almost entirely on my secretary, Miss Sinzininex.

HERBERT J. PATERSON.

February 1914.

PREFACE

IN the following pages I have attempted to give a practical account of " The Diagnosis and Treatment of those Affections of the Stomach which are amenable to Direct Surgical Interference." [1] The significance of the information to be gained from " test-meals " is considered in some detail, because I believe that such investigations are of great value in the diagnosis of gastric disease. It appears to me strange, that whereas at the present time, bacteriological and pathological examinations are used so extensively in the diagnosis of disease, chemical methods are so rarely made use of in practice, and dealt with so briefly in text-books. The stomach-tube should be part of the armamentarium of every practitioner. It is not essential that he should perform all the quantitative analyses to which I refer, but I venture to think that he would be repaid amply, if he carried out the ordinary qualitative tests, and the estimation of the total acidity of the gastric contents. The practitioner, be he consultant or otherwise, who, without the aid which such investigations afford, continues to treat a patient suffering from persistent indigestion, or gastric pain, is acting unfairly to his patient and unjustly to himself.

The advances made in recent years by the many brilliant workers in the field of gastric disease, have led to successes which are among the greatest achievements of surgery. Lives are saved and suffering is relieved to an extent undreamt of twenty years ago. But much remains to be

[1] The title of the essay for which the Jacksonian Prize (R.C.S.) 1904 was awarded to the author.

done. The loss of human life from cancer casts a shadow
over the triumphs of the past. The saddest and most dis-
couraging part of the work of a surgeon is the frequency
with which he sees patients suffering from cancer so ad-
vanced, that the time for a radical operation has gone for
ever. Especially is this true of gastric cancer. In less than
5 per cent of the cases I see, is a radical operation possible.
The stomach is affected with cancer more frequently than
any other organ of the body, and yet, partial gastrectomy is
one of the uncommon operations of surgery. This reproach
to our profession would be removed, in some measure at
least, were the use of the stomach-tube part of the routine
of gastric diagnosis. On this point I feel strongly, and have
no hesitation in saying, that the number of cases of gastric
cancer treated successfully by operation, ought to be, and
could be, increased greatly, if only the cases were recognised
earlier. Gastric cancer, contrary to popular belief, *can* be
treated successfully, and earlier diagnosis *is* possible, even
with the means at present at our disposal.

The operation of gastro-jejunostomy is not a panacea for
all the ills to which the stomach is heir, but when performed
in cases in which there is a definite organic lesion of the
stomach or duodenum, the results are excellent. It is the
keystone of gastric surgery.

It has been my aim to present methods of diagnosis
and treatment, in a manner which may prove useful in
practice to practitioners and students. If in any degree this
aim be realised, the time spent by the reader and the author
will not have been wasted.

Limitations of space have prevented me from referring
to all the views and operative methods of those who have
done such admirable work in gastric surgery. While the
opinions expressed are based largely on the experience gained
from my own failures and successes, doubtless the sub-

conscious mind is influenced, even more than one realises, by the fruits of the labour of others. It is, therefore, a sincere pleasure to acknowledge the stimulus and inspiration I have derived from perusal of the writings and witnessing the work of many brilliant exponents of our art, especially Dr. Soltau Fenwick, Sir Arbuthnot Lane, Mr. Rutherford Morison, Sir Berkeley Moynihan, Mr. Harold J. Stiles and Sir J. Bland Sutton in this country, and Drs. W. J. and C. H. Mayo, Dr. J. B. Murphy, Dr. John B. Deaver, Dr. Howard Kelly, and Professor Kocher abroad. With Dr. Fenwick it has been my privilege to be associated during the past eleven years, and it was through him that my interest in gastric surgery was begun and fostered.

I should be indeed ungrateful, if I did not put on record how much I owe to my former teacher, Mr. Harrison Cripps. While I was his house surgeon at St. Bartholomew's and his assistant in private, I had the advantage of helping him in his abdominal operations. The lessons I learnt have been of inestimable value to me in my subsequent work.

To my friend, Mr. Chas. Gibbs, I am indebted for many valuable suggestions, for which I offer him my thanks.

I have also to thank Dr. Fenwick, Dr. Porter Parkinson, and Messrs. Allen and Hanburys for the loan of blocks.

I have been fortunate in my helpers. To my former secretary, Miss Rendle, I am greatly indebted for much help during the preparation of the earlier portions of the work.

Last, but not least, my secretary, Miss Sinzininex, in addition to preparing the index, and sharing with me the correction of the proofs, has given to me constant and valuable assistance at all stages of my task, and I desire to express to her my sincere thanks.

HERBERT J. PATERSON.

9 Upper Wimpole Street, W.
January 1913.

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CHAPTER I

THE METHOD OF INVESTIGATION OF GASTRIC CASES

THE object of this chapter is to give an outline of the method to be pursued in investigating a case of gastric disease, together with a brief summary of the main points of diagnostic importance. For fuller details as to symptoms and diagnosis, the reader is referred to the various chapters dealing with particular lesions.

1. THE HISTORY

From a diagnostic point of view the history is of the utmost importance, and great care should be taken to obtain an accurate and exact account of the origin and progress of the patient's illness. The histories recorded in hospital notes usually are so vague and incomplete that they are valueless. Students nowadays are so obsessed with the importance of blood counts, X-ray examinations, &c., that they are apt to relegate even physical examination to a second place. Laboratory investigations are an aid—and a valuable aid—not a substitute for clinical work. No chemical or pathological researches can do away with the necessity for an exact history of the case and a careful clinical examination.

To obtain an accurate history requires much patience and detailed questioning. The patient is apt to describe

A

the symptoms of the present, and to forget the symptoms of
the past. For example, a patient may state that his pain
is almost constant; further cross-examination, however, may
bring out the truth that at first the pain was not constant,
but came on from two to three hours after food. In the
later stages the constancy of the pain so impresses the
patient, that the memory of the earlier sequence of events
is almost obliterated.

Pain is usually a prominent symptom in gastric disease.
The date of the first onset of the pain and its situation must
be noted. The probability is that pain to the right of the
middle line is duodenal or biliary, pain to the left, gastric.
In the early stages the pain of any of these conditions may
be referred to the epigastrium. Usually the pain of appen-
dicular gastralgia is situated in the epigastrium, and may
radiate to the right iliac region. Gall-bladder pain may ·
radiate to the costal margin, right shoulder, or dorsal region.
Radiation in gastric or duodenal ulcer is less common, and
when present may indicate invasion of the pancreas. In
perforated duodenal ulcer the patient may refer the pain to
the right iliac fossa, but further questioning will elicit the
fact that at first the pain was limited to the upper abdomen.

Inquiry should be made into the **character of the pain.**
The pain of biliary colic may be agonising in its intensity;
the pain of duodenal ulcer is rarely of such severity, although
in some instances patients may be " doubled up " during an
attack. In appendicular gastralgia often the patient speaks
of severe discomfort rather than of pain.

The duration of the pain, its relation to food, and the
extent to which it is influenced by drugs are important.
Gall-bladder pain may be transient, or may last for many
hours, and is uninfluenced by food or alkalis. Duodenal or
gastric ulcer pain increases in intensity towards the time of
the next meal, and is eased by taking more food, or by the

administration of alkalis. The pain of carcinoma is less severe, and is less influenced by food or by alkalis. In gastric ulcer the patient is hungry but does not eat for fear of the pain, in carcinoma the patient does not eat because he has no appetite, and frequently he has marked aversion from all forms of meat. The pain of biliary colic is capricious in its onset, and follows no definite sequence. The attack commences without apparent cause. In duodenal or gastric ulcer often the patient can trace the onset of an attack to a sudden chill, to some indiscretion in diet, or to mental worry. The pain of appendicular gastralgia may be brought on by severe physical exercise. In duodenal ulcer the sequence of events is remarkably constant ; the pain, coming on two or three hours after food, increasing in intensity, and relieved by food, often forms a clinical picture which cannot be mistaken.

In hypersecretion, whether due to duodenal or to gastric ulcer, or to appendicular disease, a very constant feature is the onset of pain in the early hours of the morning, waking the patient from sleep.

The kinds of food which cause pain should be inquired into ; in gastric and duodenal ulcer the patient feels more comfortable on a milk diet, in appendicular gastralgia milk diet has not the same effect in lessening the pain. In gallbladder trouble change of diet has no effect, in gastroptosis and atony of the stomach fluids cause more discomfort than solids.

In appendicular gastralgia the pain comes on either immediately or shortly after food. The same is true to a less extent in gastric ulcer towards the cardiac end of the stomach. As a rule, the longer the interval between the taking of food and the onset of the pain, the greater the probability of the ulcer being in the duodenum. The constancy, or otherwise, of the pain is often suggestive ; pain,

definitely related to food, coming on in attacks, with freedom
from symptoms for weeks or months, generally spells duo-
denal ulcer. Similar attacks with discomfort and flatulence
in the intervals may indicate duodenal ulcer or appendicular
gastralgia. Irregular, short, severe attacks of pain, not
influenced by food, suggest gall-bladder disease. Constant
pain may accompany gastric cancer or hour-glass stomach.
When such pain succeeds definite attacks with free intervals,
it is indicative that stenosis, from ulcer, has ensued, or that
an ulcer has invaded the peritoneum, or is invading neigh-
bouring organs.

If there be a history of vomiting, its frequency, character,
and amount should be noted. Copious vomiting, occurring
every two or three days, of material containing articles of
diet known to have been taken on previous days, indicates
pyloric stenosis. The eructation or vomiting of a highly
acid fluid without food remains, is symptomatic of hyper-
secretion.

The vomiting of bright blood in any quantity is due
probably to gastric or to duodenal ulcer, to cirrhosis of the
liver, or more rarely to aneurysm. The emesis of coffee
ground material may be due to malignant disease or ulcer.
The occurrence of " black stools " should be inquired into,
and an attempt made to determine whether they were
due to hæmorrhage or the taking of bismuth or other
drugs.

The habits of the patient, especially with reference to
over-indulgence in alcohol and cigarette smoking, may give
a clue to the origin of the gastric trouble, while the possi-
bility of syphilis must be borne in mind. A history of attacks
of jaundice points to gall-bladder disease, either as a com-
plication of gastric disorder, or as the cause of the gastric
symptoms.

2. PHYSICAL EXAMINATION

This includes both general and special examination.

A. **General.**—The general appearance of the patient should be noted. Severe anæmia in conjunction with gastric pain, in an otherwise healthy, well-nourished patient, suggests hæmorrhage from an ulcer. Anæmia with a worn-out look arouses suspicion of cancer. In cancer patients a diagnosis is often written on their faces. In duodenal ulcer the blood-pressure is often high, in carcinoma low, and the pulse feeble. The possibility that gastric pain may be due to the " gastric crises " of locomotor ataxy must not be forgotten, and the patient therefore should be examined for signs of this disease.

Kidney trouble, too, may simulate gastric disease, hence the need for a careful examination of the urine. A pelvic examination should be part of the routine, at any rate before deciding on any operative procedure.

B. **Special.**—Inspection of the abdomen may reveal a tumour, or an obviously dilated stomach. If there be visible waves of gastric peristalsis, pyloric stenosis is present. A dilated stomach without visible peristalsis may be due to gastric atony.

By palpation a pyloric tumour, or a swelling in the region of the gall-bladder, may be felt. A succussion splash felt more than three hours after food suggests gastric atony.

Palpation should be practised both before and after distension of the stomach with air by means of an inflating bellows.

In this way an otherwise impalpable tumour sometimes may be felt, and a tumour of the transverse colon or liver can be differentiated from a pyloric tumour.

Inflation of the stomach will reveal gastroptosis, as the displaced upper border of the stomach can be seen plainly, sometimes as low as the umbilicus.

Auscultation is sometimes of service in the diagnosis of œsophageal stricture, or of cancer at the cardiac orifice of the stomach. Dr. Ogston has pointed out that on listening at a spot about three inches below the angle of the left scapula, an amphoric rushing sound is heard when the fluid enters the stomach. In œsophageal stricture the sound may be delayed until fourteen or sixteen seconds after the swallowing of food.

If stricture be suspected, an œsophageal bougie should be passed, if necessary an anæsthetic being given to exclude spasm. Sometimes a subphrenic abscess containing gas may be diagnosed by means of auscultation.

Personally, I do not place much reliance on percussion or auscultatory percussion of the stomach, except in combination with distension of the viscus with air. The stomach limits mapped out by simple percussion rarely are confirmed on the operation table, or by X-ray examination.

3. THE MOTOR FUNCTIONS OF THE STOMACH

The simplest and most useful method of investigating the motor functions of the stomach is the following : The last thing at night, the stomach is washed out with warm water until the washings are returned clear. Usually about two pints of water are used. The quantity should be measured carefully, in order to ascertain whether all of it be recovered, or whether some of it be retained in the stomach. If a considerable amount of the water used be not recovered, it may be because it has passed into the distal compartment of an hour-glass stomach. After completion of the lavage, twelve ounces (340 c.c.) of milk is given to the patient. On the following morning, exactly ten hours later, the stomach tube is passed, and any fluid present in the stomach is withdrawn by aspiration with Senoran's apparatus (see Appendix, p. 320). Normally,

under these conditions the stomach should be empty. Should there be any fluid withdrawn, it is measured, and the amount noted. It is then tested for acidity, and for the presence of bile and blood. The presence of a clear acid fluid without food residue indicates hypersecretion. The presence of food remains is a sign of pyloric stenosis, or motor insufficiency. It is a useful plan to give some raisins with the milk, as the presence of the skins and pips is recognised easily in the gastric contents.

In pyloric stenosis as much as 400 c.c. of fluid containing much food residue may be recovered from the stomach.

In duodenal ulcer, without stenosis, the motor power of the stomach is good, and no food remains are present in the early morning. Frequently in duodenal ulcer there is hypersecretion, so that from 30 c.c. to 50 c.c. of fluid may be withdrawn in the early morning. It is a clear or opalescent yellowish fluid, containing no food residue, and having an acid reaction. In appendicular gastralgia hypersecretion is a frequent, and in gastric ulcer an occasional symptom.

4. EXAMINATION OF VOMIT

When vomiting is one of the symptoms, the amount vomited at a time should be carefully measured. As a rule, in dilated stomach due to atony or pyloric stenosis, vomiting does not occur more than once in the twenty-four hours, sometimes less frequently, but the quantity vomited may be enormous. In uncomplicated gastric or duodenal ulcer vomiting, when it occurs, usually does so an hour or two after meals, and consists of partially digested food. In pyloric stenosis the vomit has a very acid smell; in carcinoma it has often a very foul odour.

The vomit should be examined microscopically for blood and for bacilli, and also tested chemically for blood. Pus

in the vomit may be due to the rupture into the stomach, of
a perigastric abscess.

5. GASTRIC ANALYSIS

Personally, I attach great importance to the information
to be gained from the examination of test-meals. I need
hardly say that I do not maintain that a diagnosis should be
based solely on a chemical examination, but if the results
be interpreted in the light of the clinical history, careful
examination of the motor activity, and of the secretions of
the stomach, is a great help. In the following account I
will attempt, in general terms, to indicate the variations
found in different diseases, but it must be understood that
the results are not absolutely constant. It is impossible to
lay down hard and fast rules as to the relation of the different
lesions found to the chemical alteration of the gastric con-
tents. With experience, however, one is able to recognise
the types of analytical results which usually are associated
with particular lesions.

For purposes of gastric analysis a " test-meal " is given,
and the contents of the stomach drawn off after a given
interval. Pawlow has shown that the secretion of gastric juice
varies according to the kind of food given, and according
to the length of time digestion has progressed. Hence to
make an accurate comparison of the analysis of one test-
meal with that of another, the stomach must be empty at
the time of the administration of the test-meal, the amount
and composition of the meal must be the same, and the
time which it is allowed to remain in the stomach must be
the same. If these conditions be not complied with, the
resulting analyses are of no value. Various test-meals are
employed, but the following modification of that of Ewald
is the simplest and most generally useful.

Usually the test-meal is given in the morning, half an hour

after the investigation into the motor functions of the stomach (see p. 6) has been completed. The technique is as follows :

The test-meal I use consists of two slices of dry toast and fifteen ounces (430 c.c.) of weak tea with sugar, but no milk. The stomach contents should be drawn off exactly one hour after the patient has begun the meal.

The amount of gastric contents recovered should be measured carefully. In a normal stomach the amount recovered varies from 100 c.c. to 140 c.c. In pyloric stenosis the quantity recovered may be as much as 500 c.c. or 600 c.c. In duodenal ulcer the evacuation of the stomach is often quicker than normal, so that the amount of gastric contents recovered after a test-meal is less than in health. The colour, smell, and character of the contents are noted. The colour is usually a light brown ; if much blood be present the colour is dark brown, the so-called " coffee grounds " appearance. Sometimes bright blood, recognisable as such, is present ; in small quantity this may be due to trauma caused by the tube, in large quantity it may be from an open ulcer. Usually the smell of the gastric contents is not objectionable. If there be stagnation, the smell is very acrid, owing to the presence of butyric acid ; in malignant disease in which there is extreme ulceration or sloughing of the growth, the smell may be extremely putrid. In normal digestion the toast is finely divided ; in chronic gastritis, whether due to gastric ulcer or malignant disease, or secondary to appendix trouble, the gastric contents may be very thick and difficult to pour, and lumps of undigested toast, with much thick, ropy mucus, are present.

The gastric contents are then tested [1] qualitatively and quantitatively.

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[1] An account of the various tests and methods of analysis will be found in the Appendix.

(a) Qualitative

The qualitative tests are carried out on some of the gastric contents which have been filtered. The reaction should be acid. In severe chronic gastritis the reaction may be alkaline. The presence or absence of free hydrochloric acid is tested by Gunzberg's reagent or by the dimethyl-amido-azo-benzol test.

Free hydrochloric acid usually is present in duodenal ulcer, in gastric ulcer near the pylorus, and in gall-bladder disease, and sometimes in appendicular gastralgia.

It is absent as a rule in malignant disease, in severe chronic gastritis, in *old* gastric ulcer situated in the body or cardiac end of the stomach, in cases of hour-glass stomach, and in appendicular gastralgia.

The presence of **bile pigment** is tested for by Gmelin's test. Not infrequently in duodenal ulcer is there regurgitation of bile in sufficient quantity to give the reaction.

Volatile acids usually can be detected by smell; the chemical test is as follows : Some of the unfiltered gastric contents are placed in a test-tube, a piece of blue litmus paper, moistened with distilled water, is stretched on the mouth of the tube, and the contents heated. If volatile acids be present, the blue paper is reddened owing to the liberation of volatile acids.

Lactic acid in small quantities is present almost invariably in the gastric contents obtained after an ordinary test-meal. Its presence is of clinical significance only if it be found after the administration of a special test-meal. The procedure devised by Boas is as follows :

The stomach is washed out overnight until the water returns absolutely clear. On the following morning the stomach is washed out again and a meal of oatmeal soup is given. The soup consists of one teaspoonful of rolled oats

and 1000 c.c. (35 oz.) of water, boiled down to 500 c.c. (17 oz.), to which is added a little milk, but *no salt*. Two hours later the stomach-tube is passed, and the gastric contents drawn off. The contents are filtered, and tested for lactic acid with Uffelmann's reagent. Under these conditions lactic acid rarely is present except in gastric carcinoma. A positive test therefore is of great clinical significance, and points to cancer of the stomach.

(b) Quantitative

(i) **Total Acidity.**—In a healthy stomach the total acidity varies between 55 and 65. If the total acidity be over 70, a condition is present which may be termed hyper-acidity; if below 50, hypo-acidity.

Hyper-acidity must be distinguished clearly from hyper-chlorhydria. The total acidity as estimated by Ewald's method represents the amount of total acids in 100 c.c. of gastric contents, *i.e.* the amount of hydrochloric acid, volatile acids, and of acid phosphates. Thus hyper-acidity may be due to excess of volatile acids, with normal or slight excess of hydrochloric acid; to increased free hydrochloric acid with normal or diminished volatile acids; or on the other hand, there may be hyperchlorhydria or excess of free hydrochloric acid, with only slight hyper-acidity.

The highest total acidity I have found is 170, in a case of gastric ulcer near the pylorus, and the lowest 12, in a case of cancer of the stomach.

In duodenal ulcer some degree of hyper-acidity is the rule (in 75 per cent. of the cases), hypo-acidity the exception, the average total acidity in my series of cases being 79.

In gastric ulcer the amount of the total acidity varies considerably. In ulcer near the pylorus it is usually higher than is normal. In hour-glass stomach the total acidity is low. In gastric ulcer in the body of the stomach or at the

cardiac end, there is occasionally hyper-acidity, but more often hypo-acidity.

In malignant disease the total acidity almost invariably is low; only twice have I observed it higher than 50. The lowest total acidity I have met with is 12. Usually, it varies between 25 and 40, the average in my cases being 30. In appendicular gastralgia the total acidity is generally slightly higher than normal. The average total acidity in my series of cases of appendicular gastralgia is 65.

In diseases of the gall-bladder, as a rule, the total acidity is not markedly altered.

(ii) **Volatile Acids.**—Normally the amount of volatile acids as estimated by Harley's method varies from two to five. In appendicular gastralgia an appreciable increase in the amount of the volatile acids is the rule. On one occasion I found a percentage of 18·0. In malignant disease there usually is an increase. In one of my cases of carcinoma, confirmed by operation, there were no volatile acids present, but the growth was at the cardiac end of the stomach. In duodenal ulcer increase of the volatile acids is the exception, not the rule. In gastric ulcer, in my experience the volatile acids are not increased markedly unless there be associated appendicular disease.

(iii) **The Total Chlorides.**—In health the amount of the total chlorides varies from 0·310 to 0·330, the average being about 0·320.

In duodenal ulcer there is a very marked increase of the total chlorides; in one of my cases the amount was 0·514, the average in my series being 0·380.

When pyloric obstruction exists, often there is a decrease. In gastric ulcer there is usually an increase, less marked, but to some degree varying with the site of the ulcer. When the ulcer is near the pylorus the increase is more marked than when it is situated at the cardiac end.

When the ulcer, by cicatrisation, has produced pyloric stenosis or hour-glass stomach, commonly there is a decrease of the total chlorides.

In appendicular gastralgia the variation from the normal is not great, the tendency being towards a decrease.

(iv) **Free Hydrochloric Acid.**—In a healthy individual the amount of free hydrochloric acid in the gastric contents after a test-meal varies from 0·018 to 0·022, the average being 0·020.

In duodenal ulcer, at any rate in the earlier stage, hyperchlorhydria is the rule. When obstruction has supervened, or if the patient be examined between the attacks, especially if he has been on a purely milk diet, the free hydrochloric acid may be greatly diminished, or even absent. This, however, is the exception ; in the majority of cases, the free hydrochloric acid is increased to a marked degree. In one case under my care the amount of free hydrochloric acid was 0·127 (verified by several analyses).

In the early stages of gastric ulcer free hydrochloric acid always is present in excess, but this does not hold good for the later stages. In active ulcers near the pylorus hyperchlorhydria is the rule. Thus in a case of gastric ulcer situated close to the pylorus, 0·072 of free hydrochloric acid was present.

When a gastric ulcer has caused pyloric stenosis the free hydrochloric acid is diminished or even absent. In ulcer of long standing situated in the body of the stomach, free hydrochloric acid is diminished or absent. Invariably, in my experience, in cases where cicatrisation has caused hour-glass stomach, free hydrochloric acid is absent. In chronic gastritis free hydrochloric acid is diminished greatly, or is absent. In malignant disease free hydrochloric acid is absent almost invariably. I have met with only two instances of gastric carcinoma, verified by autopsy or by

operation, in which free hydrochloric acid was present in the gastric contents.

In appendicular gastralgia the free hydrochloric acid is diminished markedly or absent altogether, but very occasionally there is hyperchlorhydria.

(v) **The Protein Hydrochloric Acid.**—As a rule the amount of protein hydrochloric acid varies from 0·220 to 0·250. The importance of estimating the protein hydrochloric acid lies in the circumstance, that in malignant disease the amount of protein hydrochloric acid is diminished to marked degree. This is a most valuable aid in the diagnosis. In only two cases of carcinoma have I found the amount of protein hydrochloric acid to be over 0·200, and usually it is less than 0·100.

In chronic gastritis the protein hydrochloric acid is diminished. In other gastric conditions the variations are not of clinical significance.

(vi) **The Mineral Chlorides.**—In malignant disease there is almost constantly a marked increase in the mineral chlorides. As the total chlorides are diminished, it is probable that some of the hydrochloric acid is neutralised by an alkaline fluid secreted by the growth and so converted into fixed chloride.

The following are some typical gastric analyses. In all the cases to which they refer, a correct diagnosis was made and verified by operation :

Lesion.	Total Acidity.	Total Chlorides.	Free HCl.	Protein HCl.	Mineral Chlorides.
Duodenal ulcer	82	0·350	0·038	0·229	0·080
,, ,, ...	80	0·405	0·055	0·275	0·075
Gastric ulcer (close to pylorus) .	75	0·325	0·040	0·205	0·080
,, ,, (about middle) . .	78	0·308	0·025	0 206	0·077
,, ,, (hour-glass stomach)	70	0·332	0·000	0·230	0·098
Carcinoma	28	0·178	0·000	0·051	0·124
Appendicular gastralgia . .	61	0·288	0·007	0·201	0·080

6. X-RAYS

To examine the stomach by means of the X-rays, a meal is administered consisting of two ounces of bismuth oxychloride mixed with bread and milk or with porridge. The examination should be made both in the vertical and in the horizontal positions. The presence of such foreign bodies, as are opaque to the X-rays, is detected at once by examination with the screen.

The existence of an œsophageal stricture is ascertained readily, the obstruction to the onward passage of the food and the dilatation of the œsophagus above the stricture, being clearly visible. The size, shape, and position of the stomach can be determined with fair accuracy. Hour-glass stomach can be recognised by means of the X-rays, but care must be taken not to mistake a functional contraction. In organic hour-glass stomach it will be seen that the constriction does not pass from the most dependent part of the proximal compartment, as is the case in the functional condition, and further, the spasmodic contraction will disappear on vigorous massaging of the abdomen.

Spasm near the middle of the stomach is often a sign of gastric ulcer. In a dilated stomach due to pyloric stenosis, vigorous waves of peristalsis are seen, but yet very little or no food is seen to pass into the duodenum.

Occasionally a reverse peristalsis is visible. In dilated stomach due to atony, the feebleness of the waves, and the sinking of the food to the most dependent part of the stomach, indicate the cause of the dilatation.

In cases of duodenal ulcer, without obstruction, the stomach often is seen to be actively contracted (hypertonic). When examined with the X-rays in the erect position, it is apparent that the greater curvature does not reach to the umbilicus. In gastric ulcer hypertonus rarely is present.

7. GASTROSCOPE

At present the gastroscope is not of great assistance in the diagnosis of gastric disease. An ulcer may be seen, but it is impossible to differentiate between a simple lesion and a malignant one. The best instrument is that designed by Dr. William Hill.

8. EXPLORATORY INCISION

When all other means of diagnosis have failed, abdominal exploration may be indicated. This rarely is necessary except in cases of suspected cancer. In the great majority of cases a diagnosis of the necessity for surgical treatment can be made, but if cancer of the stomach is to be dealt with surgically, with good result, the abdomen must be opened, in some cases, without a definite diagnosis having been made.

CHAPTER II

THE PREPARATION AND TREATMENT OF PATIENTS BEFORE GASTRIC OPERATIONS

LIKE success in other walks of life, success in gastric surgery depends on attention to detail. There are some who scoff at elaborate preparations, minimise the importance of after-treatment, and regard the patient's fate as sealed for good or ill when the wound is closed. I think this is a mistake. Attention to detail in the treatment of patients before and after operation, is second only to the skilful performance of the operation itself. Often skilful nursing and after-treatment will pull through a seemingly hopeless case, and always will add greatly to the patient's comfort and prospect of a speedy convalescence. There is no finality in surgery. Methods are being changed continually. I do not propose to describe, or even to summarise, all the different methods in use, but simply to give an account of those, which from an experience of over a thousand operations, as well as from watching the practice of many other surgeons in this country, America, and on the Continent, personally I have found to give the best results, and which from experience I can recommend.

PREPARATORY TREATMENT

The minimum period of preparation for an abdominal operation (except, of course, in case of emergency) should be three days, but whenever practicable I prefer the preparation of the patient to be commenced a week before the date fixed for the operation. During this period the bowels are

well opened by several doses of calomel, the last dose being given two nights previous to the operation. On the morning of the day before operation a dose of castor oil is given. The doses of these drugs must be regulated according to the age and strength of the patient, so as to secure thorough emptying of the bowels without purging. Diarrhœa is a bad preparation for an abdominal section. Enemata may be necessary as well as calomel, and in any case a plain soap-and-water enema should be given on the morning of the operation. It is a good plan for the patient to try sleeping in the propped-up, semi-sitting position, otherwise this may be found irksome after the operation. Likewise the patient should be encouraged to practise taking deep breaths at intervals during the day. This will tend to minimise the danger of hypostatic congestion of the lungs after the operation. I think, too, that such preparations give the patient something to think about, and give confidence by encouraging the belief that everything possible is being done to ensure a successful operation. During the whole period of preparation the patient is kept on a fluid diet, and for at least fifty hours nothing but sterilised milk and tea is allowed. By this means it is possible, as Professor Harvey Cushing has shown, to render sterile, the upper part at least, of the alimentary tract.

Especial care should be given to the condition of the mouth. The teeth should be brushed three times daily with carbolic tooth powder, and the mouth rinsed frequently with listerine or hydrogen peroxide (6 volume solution). Stumps or badly-decayed teeth should be extracted.

I have no doubt that some cases of sepsis after abdominal operations are due to a septic state of the mouth. Cleansing the mouth, and emptying of the intestinal canal, greatly reduce the risk of infection from within. The patient should remain in bed for two days previous to the operation.

Preparation of the Operation Area.—The skin of the abdomen is prepared in the following way : Two nights previous to the operation the pubes is shaved, and the patient takes a hot bath. If the patient be too ill for a bath, the abdomen is washed over with acetone. On the night previous to the operation the *whole* abdomen is well rubbed over for five minutes with acetone, and then swabbed over with iodine paint. This paint is made up as follows :—

Liq. Iodi Fort.	℥i.
Spt. Vini Recti	℥ii.
Aq. Destill.	℥i.

Then the whole abdomen is covered with a dry sterilised sheet of gauze or lint, which is kept in place by a sterilised bandage. As soon as the patient is placed on the operation table the bandage is removed, and while the patient is being anæsthetised the whole abdomen is painted over again with iodine paint. When anæsthetisation is complete, the super-fluous iodine is wiped off with a piece of sterile gauze. After an extensive trial of many other methods of skin prepara-tion, I have found that the one above described is not only the simplest, but the most reliable. It takes little time, there is no necessity (except in exceptional cases) to shave the whole operation area, and the long exposure necessitated by most other methods is avoided. The skin tested bacterio-logically is invariably sterile. In a number of cases I have snipped off a fragment of skin at the conclusion of the operation and placed it in a culture tube, and in one case only has a growth of micro-organisms resulted on incubation. Further—and this to my mind is a great advantage of this method—its rapidity and reliability render it equally appli-cable for emergency operations. Simplification and uni-formity of methods form one of the ideals to be aimed at in all surgical procedures.

In cases of pyloric and intestinal obstruction the stomach

should be washed out two or three hours before the operation. In elderly and feeble patients it is a good plan to give a pint of saline solution per rectum a couple of hours before the operation. As an additional precaution in such cases, while the patient is being anæsthetised, a needle may be inserted in the axilla, and a pint of saline solution run into it from a rubber bag, as practised by Sir Arbuthnot Lane.

During a visit to the chief clinics of the United States in the year 1907, I was impressed greatly with the method of open administration of ether employed so extensively in that country, and since then have had scarcely any other anæsthetic given to my patients, and I am convinced, contrary to my former opinion, that ether is a far more satisfactory anæsthetic than chloroform. Ether is undoubtedly safer, and is not less pleasant than chloroform ; it is followed very rarely by post-anæsthetic vomiting, and is less depressing than chloroform. With ether the signs of shock are much less marked than with chloroform.

If the patient is to have ether by the open method, a hypodermic injection of morphia, gr. $\frac{1}{6}$, and atropine sulph. gr. $\frac{1}{100}$, should be given half an hour before the time fixed for the operation.

The previous administration of morphia composes the patient before operation, diminishes the amount of ether requisite to induce and maintain anæsthesia, and delays the return to consciousness of pain. The atropine checks troublesome secretion of mucus in the trachea and bronchi, and lessens the sweating so common when ether is given alone.

The work of Dr. G. W. Crile on anocci-association opens up a new vista in abdominal surgery, but as yet I have not had sufficient experience of it to enable me to express an opinion on its uses and limitations.

CHAPTER III

OPERATIONS ON THE STOMACH

The Incision.—It is best to open the abdomen just to the right of the middle line. A gastro-jejunostomy *can* be performed through an incision 2½ inches in length, but such an incision does not suffice for an adequate examination of the gall-bladder and appendix. No operation on the stomach should be performed until the whole of the viscus has been examined, and the surgeon has satisfied himself as to the lesion present. Without adequate exposure it is easy to overlook an hour-glass stomach, and to mistake the distal pouch for the whole of the viscus. If, as has been done in some of the recorded cases, a gastro-jejunostomy be performed on the distal pouch, the patient will gain no relief. It is absolutely essential, therefore, before proceeding with any operative measure, to make a very careful examination of the stomach and other viscera. The time so spent is never wasted. In every case the appendix and gall-bladder should be examined, and dealt with, if necessary, and if the patient's condition permit. After incising the skin and fascia, the sheath of the right rectus is divided just to the right of the middle line. The rectus muscle is displaced outwards, and the posterior sheath and peritoneum divided between two pairs of forceps, in the line of the incision through the anterior sheath.

Closure of the Wound.—The closure of the wound is effected as follows: A large curved needle armed with a double thread of No. 2 iodised catgut is used. The suture

is commenced at the lower end of the wound, and after the first suture has been tied, the needle is passed from the inner surface of the peritoneum, so as to turn the cut edge of the peritoneum outward, as suggested by Dr. J. B. Murphy. The suture includes the transversalis fascia and posterior sheath of the rectus. It is continued without interruption to the upper end of the wound, where the needle is passed under the last loop and drawn tight. The needle is laid aside without cutting the catgut. I have all my needles made with a special " grip-eye " designed to prevent accidental unthreading of the suture material (Fig. 1). A few silkworm-gut sutures are now passed through the remaining structures of the abdominal wall, viz. through the skin, anterior sheath of the rectus, the fibres of the recti muscles, and anterior sheath and skin of the opposite side. These sutures are passed at intervals of an inch, so that in a wound four inches long, three interrupted sutures are employed. The ends of these sutures are

FIG. 1.—The author's secured temporarily with pressure forceps.
" grip-eyed " needle.
The anterior sheath of the rectus is now sutured with the same needle and suture as was used for the peritoneum. The suture is continued from the upper angle of the wound to the lower, where it is tied to the end which was left long for the purpose when commencing the suturing. The ends are then cut short.

The skin incision is closed by a sub-cuticular suture of silkworm-gut. The operation area is now painted over with iodine paint, and covered with silver leaf. A folded strip of gauze about an inch wide is laid lengthwise on the wound, and the interrupted sutures are tied over it. This anchors the dressing, and at the same time prevents the discomfort caused by the sutures cutting into the skin.

The important points in the technique of abdominal wounds are :

1. Perfect hæmostasis.

2. Avoidance of tearing of muscle or fascia.

3. Obliteration of " dead spaces."

4. The protection of the edges of the wound from bruising and contamination.

5. Accurate apposition of each layer.

1. GASTROTOMY

Indications :

(i) For the removal of foreign bodies from the stomach or lower end of the œsophagus.

(ii) For the removal of growths from the interior of the stomach.

(iii) For dilating the pylorus (Loreta's operation).

(iv) In rare instances for gastric hæmorrhage.

(i) For the Removal of Foreign Bodies

(a) *From the Stomach.*—All kinds of foreign bodies have been found in the stomach : hairballs, nails, false teeth, pins, stones, coins, and many other articles. Often they cause no symptoms, and in some cases have remained in the stomach for years without causing trouble.

After opening the abdomen the stomach is packed round with pads so as to prevent contamination of the peritoneal cavity with gastric contents. The stomach is drawn partially out of the abdomen and a vertical incision about two inches in length is made into the viscus. The foreign body is removed by a suitable pair of forceps. In the case of a small foreign body it is a help to pass a hand up behind the transverse colon to the posterior aspect of the stomach, and to steady the foreign body while it is being grasped by the forceps. The wound in the stomach is then sewn up with

two continuous sutures of linen thread, an inner suture including all coats of the viscus, and an outer serous suture. The abdominal incision is then closed, as described already, without drainage.

(b) *From the Lower End of the Œsophagus.*—Occasionally a foreign body lodges in the lower end of the œsophagus and neither can be drawn upwards, nor pushed downwards into the stomach. In such cases the foreign body usually can be removed by forceps introduced through a gastrotomy opening. Some years ago I saw a gentleman who, in a fit of melancholia, had swallowed a metal case $3\frac{1}{2}$ inches long containing a glass creasote inhaler. After localisation with the X-rays, I opened the stomach and withdrew the case by means of a pair of long forceps, without difficulty.

Dr. Bull removed successfully a peach stone by attaching a sponge to a piece of string and dragging the sponge upwards, from a gastrotomy opening, to the mouth.

Gastrotomy for the removal of foreign bodies is a very safe operation, the mortality rate under modern conditions being well under 2 per cent.

(ii) For the Removal of Growths from the Interior of the Stomach

In these cases, as more room is required, it is advantageous to make the incision into the stomach in the direction of its long axis. After removal of the growth and the arrest of all hæmorrhage, the stomach is closed as usual by two continuous sutures.

(iii) For Dilating the Pylorus (Loreta's Operation)

This operation seldom is performed nowadays. Gastrojejunostomy is safer, although Mr. Burghard has had very good results in the treatment of infantile pyloric stenosis by means of dilatation. After opening the stomach, a pair of dilating

forceps is introduced into the pylorus, which is then stretched as much as is possible without tearing the peritoneal coat.

(iv) For Gastric Hæmorrhage

For gastric hæmorrhage, the incision into the stomach is made in the direction of its long axis, and the source of the hæmorrhage sought for, an electro-cystoscope being used to illuminate the interior of the organ. The posterior wall of the stomach may be examined by invaginating it, and pushing it forwards through the incision in the anterior gastric wall.

Personally, I am not in favour of operations for gastric hæmorrhage during the progress of bleeding. The mortality from such operations is very high, and in my opinion it is safer to trust to medicinal means for the temporary arrest of the hæmorrhage, and then to perform gastro-jejunostomy later on, if it be considered advisable.

2. GASTROSTOMY

Indications :
(i) Malignant stricture of the œsophagus.
(ii) Impermeable fibrous stricture of the œsophagus.

Many different methods of performing gastrostomy have been devised. In my experience that of Senn is the best, and it is the one I now employ invariably. It is performed as follows :

A vertical skin incision $2\frac{1}{2}$ inches long is made over the outer third of the left rectus, commencing one inch below the costal margin. The anterior sheath of the rectus is incised and fibres of the muscle separated, care being taken not to injure any nerves. The peritoneum is then opened, and the stomach sought by two fingers introduced into the peritoneal cavity. I have seen the colon opened in

error for the stomach, but it is a mistake which should not occur.

The stomach is drawn gently into the wound, and a spot at the cardiac end, midway between the lesser and greater curvatures, is selected for the site of the opening. A small incision is made, large enough to admit a No. 12 rubber catheter, and the catheter is introduced into the cavity of the stomach for a distance of two inches, and secured by a single catgut stitch. Three (or four if sufficient stomach be available) purse-string sutures are inserted round the tube. The first suture is passed so that it surrounds the tube in the form of a circle, with its circumference at a distance of half an inch from the tube. When the suture has been inserted, the catheter is depressed by the assistant towards the cavity of the stomach, and the suture tied sufficiently tightly to grip, but not to constrict, the catheter. A second circular purse-string suture is then inserted at a distance of one quarter of an inch from the first. The catheter is again pushed inwards and the suture tightened, tied, and cut short. A third, and in some cases a fourth, suture is passed in a similar manner. When all the sutures have been inserted and tied, the result is that a cone of the stomach is inverted into the cavity of the organ, with the catheter projecting at its apex. The stomach is fixed to the parietal peritoneum by two sutures of silkworm-gut, passed through all layers of the abdominal wall and through the peritoneal and muscular coats of the stomach, at a distance of half an inch above and below the tube. The abdominal incision is closed as already described.

At the conclusion of the operation a glass funnel is attached to the catheter by means of a piece of rubber tubing with a glass connection, and half a pint of warm milk or meat essence is introduced into the stomach. The catgut securing the catheter is digested usually in nine or ten

PLATE I.—GASTROSTOMY.

FIG. 2.—The stomach has been opened, and the catheter fixed in the opening by a catgut stitch.

FIG. 3.—The first purse-string suture has been inserted and tied.   The second purse-string suture is seen ready to be tightened.

days, and the catheter comes out. It is then introduced when required for feeding purposes. Senn's method is very satisfactory. If the operation be performed properly, leakage never occurs, and the patient's condition is very different from that obtaining after operations by the older methods, after which leakage of gastric juice produces such excoriation of the surrounding skin that life is scarcely worth living.

3. PYLOROPLASTY

The operation of pyloroplasty for pyloric stenosis consists in making an incision three inches in length through the pylorus in its longitudinal axis, and then suturing the incision in a vertical direction, thus increasing the diameter of the pyloric lumen. The operation, although in principle good, is unsatisfactory in practice. The after-results are disappointing.

In my Hunterian lectures I pointed out that relapse ensues in at least 30 per cent. of the cases in which the operation has been performed. An operation which cannot show better after-results than this cannot be compared with gastro-jejunostomy. It is seldom performed in the present day.

4. PYLORO-GASTRO-DUODENOSTOMY (FINNEY'S OPERATION)

In 1902 Dr. Finney described an operation which he called a " new method of pyloroplasty," but which, strictly speaking, is a gastro-duodenostomy. It is an improvement on pyloroplasty, but whether it is an efficient alternative to gastro-jejunostomy is doubtful.

The immediate mortality, except in the hands of its inventor, is higher than that of gastro-jejunostomy, and the after-results, so far as can be determined, are hardly so satisfactory.

Dr. W. J. Mayo writes : " The Finney operation gave remarkably good ultimate results in pyloric obstruction, but in cases of unhealed ulcer existing proximal to or distal from the parts involved in the operation, less benefit was derived, unless the ulcer lay within the zone of the operative field, so that it could be coincidentally excised."

Dr. Finney's description of his operation is as follows : " Divide the adhesions binding the pylorus to the neighbouring structures ; also free as thoroughly as possible the pyloric end of the stomach, and the first portion of the duodenum.   Upon the thoroughness with which the pylorus, lower end of the stomach, and upper end of the duodenum are freed depends in a large measure the success of the operation, and the ease and rapidity of its performance.   I wish to emphasize this as one of the most important points in the operation.   Frequently, at first sight the pylorus may seem hopelessly bound down, when after a little patient toil and judicious use of the scalpel and blunt dissector it is found that it can be freed with comparative ease.   A suture, to be used as a retractor, is taken in the upper wall of the pylorus, which is then retracted upwards.   A second suture is then inserted into the anterior wall of the stomach, and a third into the anterior wall of the duodenum, at equidistant points—say about twelve centimetres from the suture just described in the pylorus.   These second sutures mark the lower ends of the gastric and duodenal incisions respectively. They should be placed as low as possible, in order that the new pylorus may be amply large.   Traction is then made upwards on the pyloric suture, and downwards in the same plane on the gastric and duodenal sutures.   This keeps the stomach and duodenal walls taut, and allows the placing of the sutures with greater facility than if the walls remained lax.   The peritoneal surfaces of the duodenum and stomach along its greater curvature are then sutured together as far

posteriorly as possible. For this row I would recommend the use of the continuous suture, as it is more easily and quickly applied, and it can be reinforced after the stomach and duodenum have been incised. After the posterior line of sutures has been placed, an anterior row of mattress sutures is taken, which are not tied, but left long, in the manner indicated. These sutures, after they have been placed, are retracted vertically in either direction from the middle of the portion included in the row of sutures. Then, after all the stitches have been placed and retracted, the incision is made in the shape of a horse-shoe. The sutures should be placed far enough apart to give ample room for the incision. The gastric arm of the incision is made through the stomach wall just inside the lowest point of the line of sutures, and is carried up to and through the pylorus, and around into the duodenum, down to the corresponding point on the duodenal side. Hæmorrhage is then stopped. It is well to excise as much as possible of the scar tissue upon either side of the incision, in order to limit, as far as possible, the subsequent contraction of the cicatrix. This procedure I carried out in two of my cases with great satisfaction, and I should strongly recommend it in all cases where the walls of the pylorus are much thickened and there is much scar tissue present. It is well, too, to trim off with scissors redundant edges of mucous membrane, in order to prevent the formation of a valve-like fold of mucous membrane at the new pylorus. A continuous catgut suture is now taken through and through all coats of the intestine on the posterior side of the incision. This reinforces the posterior line of sutures, secures better approximation of the cut edges of the mucous membrane, and prevents the reunion of the divided intestinal walls. The anterior sutures are then straightened out and tied, and the operation is complete, unless one wishes to reinforce the mattress sutures with a few Lambert stitches. This pro-

cedure, as is readily seen, gives the minimum of exposure of
infected surface.   All the stitches are placed, and the posterior
row tied, before the bowel is opened, and it remains open
just long enough to control the hæmorrhage.   The size of
the newly-formed pyloric opening is limited in this opera-
tion only by the mobility of the stomach and duodenum and
the judgment of the operator.   In all of my cases the incision
has been about twelve centimetres in length, and could have
been made longer had I chosen to make it so.   Unless the
stomach is very much dilated, or has descended to an un-
usual extent, the lower limit of the new outlet is at or near
the level of its most dependent portion."

5. GASTRO-JEJUNOSTOMY

The operation of gastro-jejunostomy consists in making
an anastomotic opening between the stomach and upper
part of the jejunum.   The communication may be made on
the anterior or on the posterior wall of the stomach.

Physiologically the anastomotic opening should be made
as near to the pylorus as possible.   In performing a pos-
terior gastro-jejunostomy it is an anatomical impossibility
to place the stoma at the pyloric end of the stomach.   In
the anterior operation, on the other hand, the stoma can
be placed close up to the pylorus, but the long loop which
is necessary to bring the jejunum over the transverse colon
is anatomically unsound.   To put the matter succinctly,
the posterior operation is anatomically good, but physio-
logically bad, the anterior operation is physiologically good,
but anatomically bad.   Some years ago I tried the plan of
making a hole in the gastro-colic omentum, then drawing
the jejunum through the aperture and anastomosing it to
the anterior surface of the stomach close to the pylorus.
I believe that a similar method has been practised by Mr.

Sherren and by Mr. Basil Hall. By this method it is possible
to place the stoma close to the pylorus, and at the same
time to avoid the long jejunal loop of the ordinary anterior
operation, but the operation is a troublesome one on account
of the vascularity of the gastro-colic omentum in the region
where the artificial aperture has to be made. In the few
cases in which I have used this method, the results have
been satisfactory, but further information is required as to
the remote results.

Nowadays the posterior operation is performed almost
universally. Although usually I perform ·the posterior
operation, I am of opinion that it is immaterial which
method be adopted. My experience is that either operation,
in suitable cases, gives excellent results. I think that per-
haps there is a slightly greater tendency to vomiting during
the first few days after the anterior operation. The patients
on whom the posterior operation is performed seem a little
more comfortable at first, and convalesce a little more rapidly.
As to the ultimate results there appears to me to be no
difference. The chief objection to the anterior operation is
the possibility, that it is more apt to be followed by a
jejunal ulcer than the posterior no-loop operation. But this
is not certain. I will describe both methods in detail.

Anterior Gastro-jejunostomy.—After opening the peri-
toneal cavity the stomach and transverse colon are drawn
gently upwards, and two fingers introduced into the abdomen
seek the commencement of the jejunum close to the left of
the spine at the root of the transverse meso-colon.

Having made certain of the origin of the jejunum, it is
traced downwards, and a portion is selected which can be
applied to the anterior wall of the stomach without con-
stricting the transverse colon. The selected portion of the
jejunum is then clamped in a long pair of forceps, the blades
of which are sheathed in rubber tubing (Plate III, Fig. 6).
The portion clamped should be at least four inches in length,
and is usually from six to eight inches from the duodeno-
jejunal junction, the distance depending on the extent of
the great omentum.

PLATE III.—ANTERIOR GASTRO-JEJUNOSTOMY.
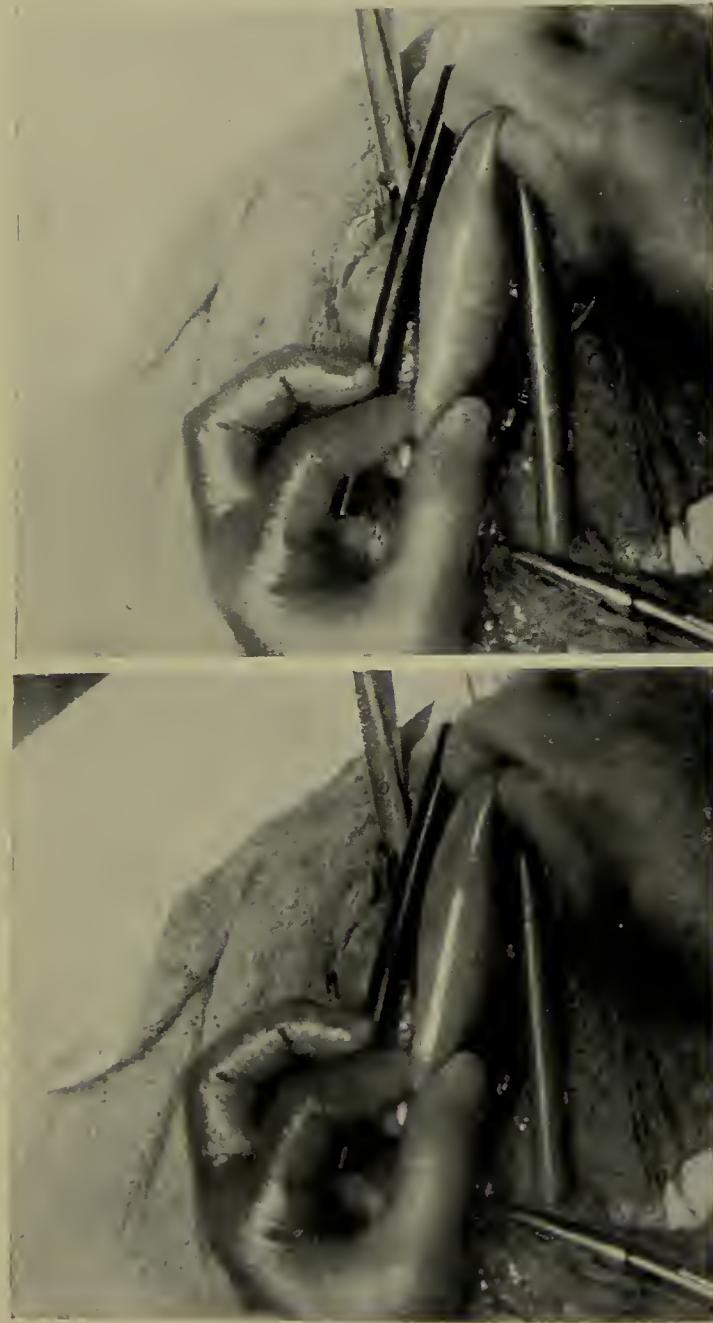
FIG. 6.—The jejunum is held by the operator while the assistant applies the clamps.

[For the photographs of gastro-jejunostomy I am indebted to Dr. Howard Kelly. They were taken by him during two of my operations while he was on a visit to this country two years ago.]

PLATE IV.—ANTERIOR GASTRO-JEJUNOSTOMY.

Fig. 7.—The jejunum clamped eight inches from its origin, which is grasped by the fingers of the left hand.

(Plate IV, Fig. 7.) The intestine has been clamped. The origin of the jejunum is shown grasped by the fingers of the left hand.

The transverse colon is drawn downwards (Plate V, Fig. 8) in order to make sure that the clamped portion of the jejunum can be applied to the stomach without tension.

PLATE V.—ANTERIOR GASTRO-JEJUNOSTOMY.

FIG. 8.—The transverse colon is drawn downwards, so as to make sure that the loop of jejunum can be applied to the stomach without tension.

PLATE VI.—ANTERIOR GASTRO-JEJUNOSTOMY.
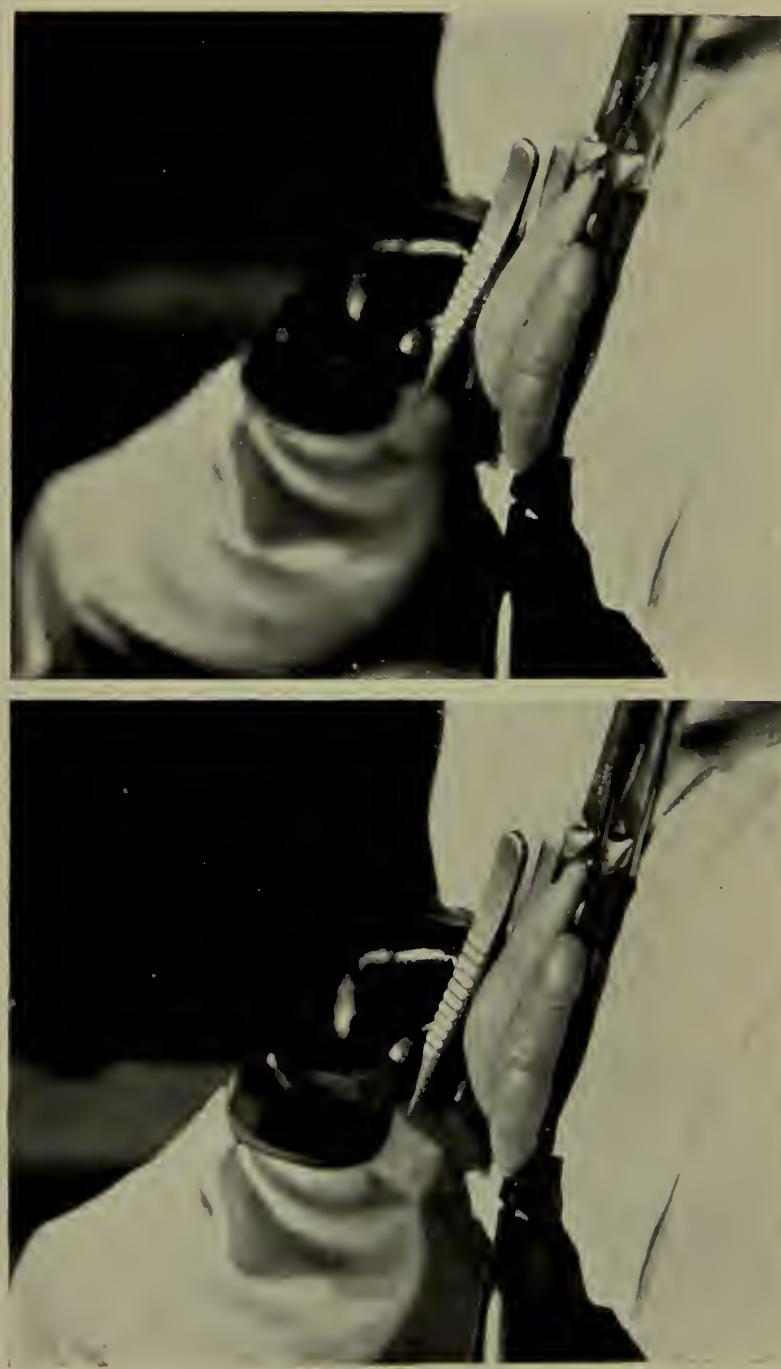
Fig. 9.—The clamped portions of viscera ready for suturing. The abdominal cavity is shut off by pads.

A portion of the anterior wall of the stomach, at least four inches in length, is clamped in a second pair of forceps. The portion clamped should be as close to the pylorus as possible, and near to the lower border of the stomach.

Pads are tucked between the clamps and on each side of them, so that no viscera are visible in the field of operation, except the required portions of stomach and jejunum.

The handles of the forceps are held side by side by the assistant, thus bringing the clamped portions of stomach and jejunum side by side (Plate VI, Fig. 9), the upper end of the clamped jejunum being on the patient's left side, and the lower end to the right. The anastomosis is thus iso-peristaltic.

A guide suture is inserted at each end of the clamped viscera (Plate VII, Fig. 10). These sutures are four inches apart, and their object is to ensure that the line of suturing shall be straight.

PLATE VII.—ANTERIOR GASTRO-JEJUNOSTOMY.

FIG. 10.—The stomach and jejunum united at each end by a guide suture.

PLATE VIII.—ANTERIOR GASTRO-JEJUNOSTOMY.

FIG. 11.—The posterior half of the serous suture has been inserted.

The anastomosis is now effected by means of two continuous sutures, an inner hæmostatic suture penetrating all the coats of the viscera, and an outer approximating suture including the peritoneal, muscular, and submucous coats. For both sutures I use a very fine black Irish linen thread (introduced by Mr. H. J. Stiles). The outer serous suture is commenced at the left end of the clamped portions of stomach and jejunum, and is continued towards the right for four inches (Plate VIII, Fig. 11). Without being unthreaded, the needle is clamped with a pair of pressure forceps and laid upon the muslin sheet above the wound, ready for use later.

A portion of the stomach as close to the suture line as possible is picked up with a pair of dissecting forceps, and the stomach is opened by cutting into it with a pair of small scissors (Plate IX, Fig. 12). The incision is then prolonged in both directions until the opening measures three inches. I attach great importance to making the anastomotic opening of sufficient size. I have a mark on the handle of my scalpel $2\frac{1}{2}$ inches from its extremity, and by using this as a measure, I make sure that the opening is at least $2\frac{1}{2}$ inches in length.

A similar opening is made in the jejunum. As the viscera are opened the mucous membrane pouts into the wound. With a pair of scissors, about a quarter of an inch of the mucous membrane is removed on each side of the opening into the stomach. As a rule I do not remove any of the jejunal mucosa.

PLATE IX.—ANTERIOR GASTRO-JEJUNOSTOMY.
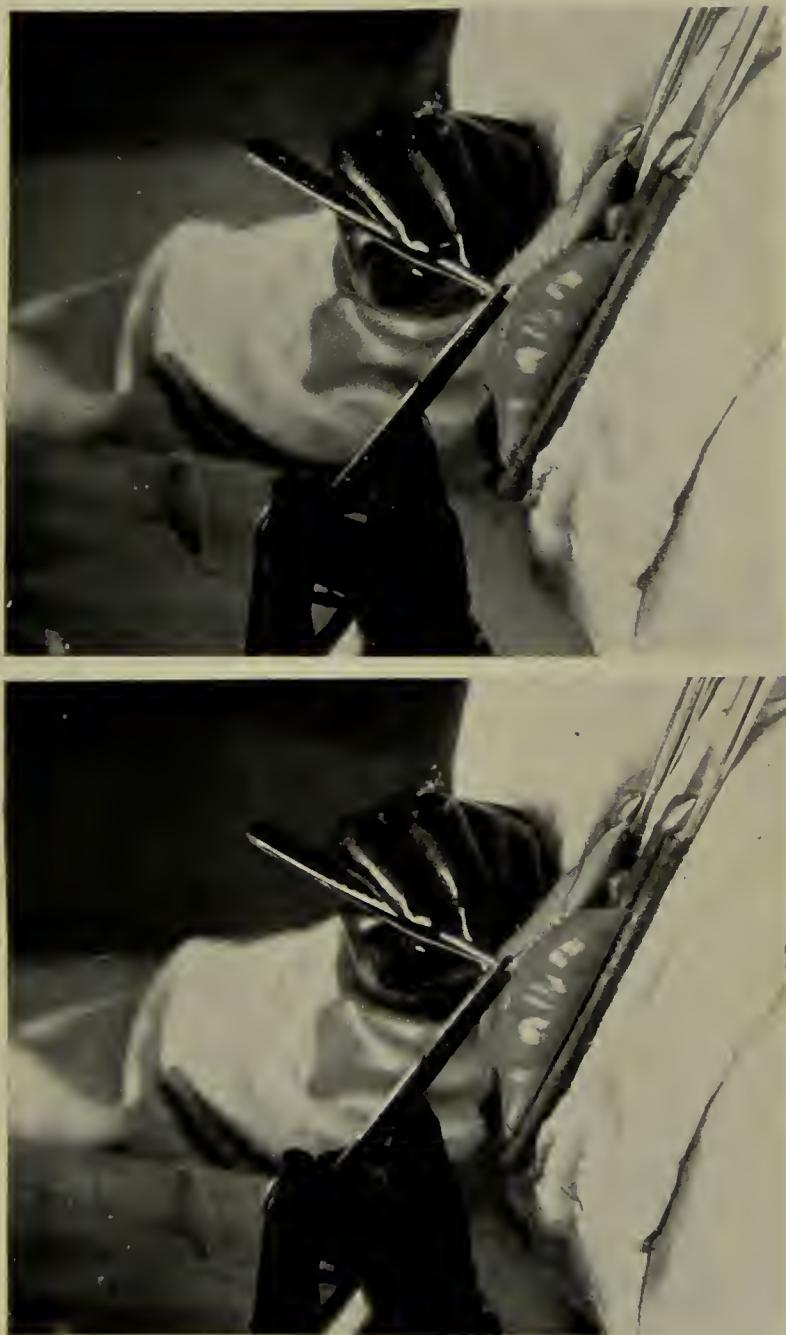
FIG. 12.—Opening the stomach.

PLATE X.—ANTERIOR GASTRO-JEJUNOSTOMY.
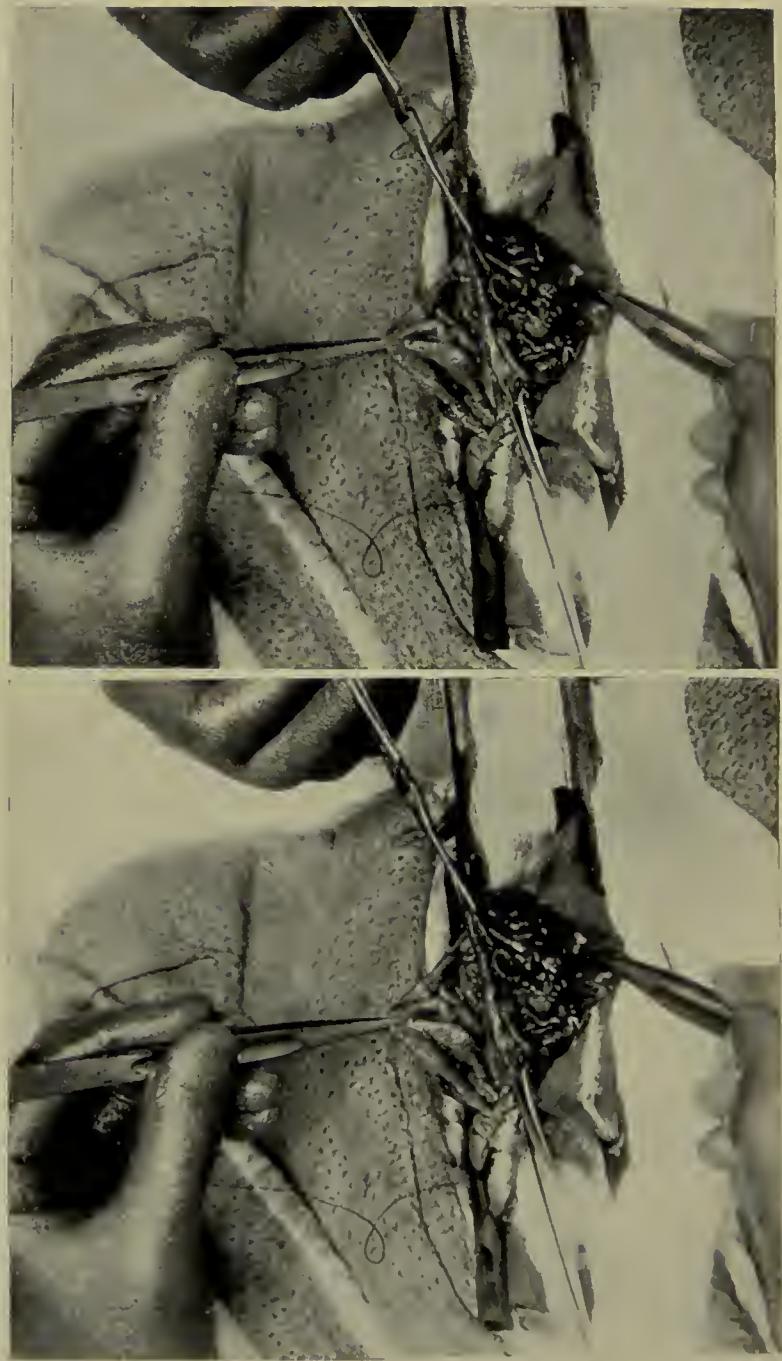
FIG. 13.—Stomach and jejunum opened. The walls of the stomach and jejunum held in apposition by two pairs of Porrier's forceps, ready for insertion of posterior layer of hæmostatic suture.

The inner or hæmostatic suture, penetrating all coats of the stomach and jejunum, is now introduced. The posterior cut edges of the walls of the stomach and jejunum are held in apposition by means of two pairs of Porrier's forceps, one pair being applied at each end of the opening (Plate X, Fig. 13).

The inner suture is commenced at the same point as the serous suture (Plate XI, Fig. 14), and is continued without interruption to the starting-point.

For the insertion of the posterior layer the needle is passed through the jejunal mucosa, through the wall of the jejunum, then through the peritoneal, muscular, and mucous coats of the stomach.
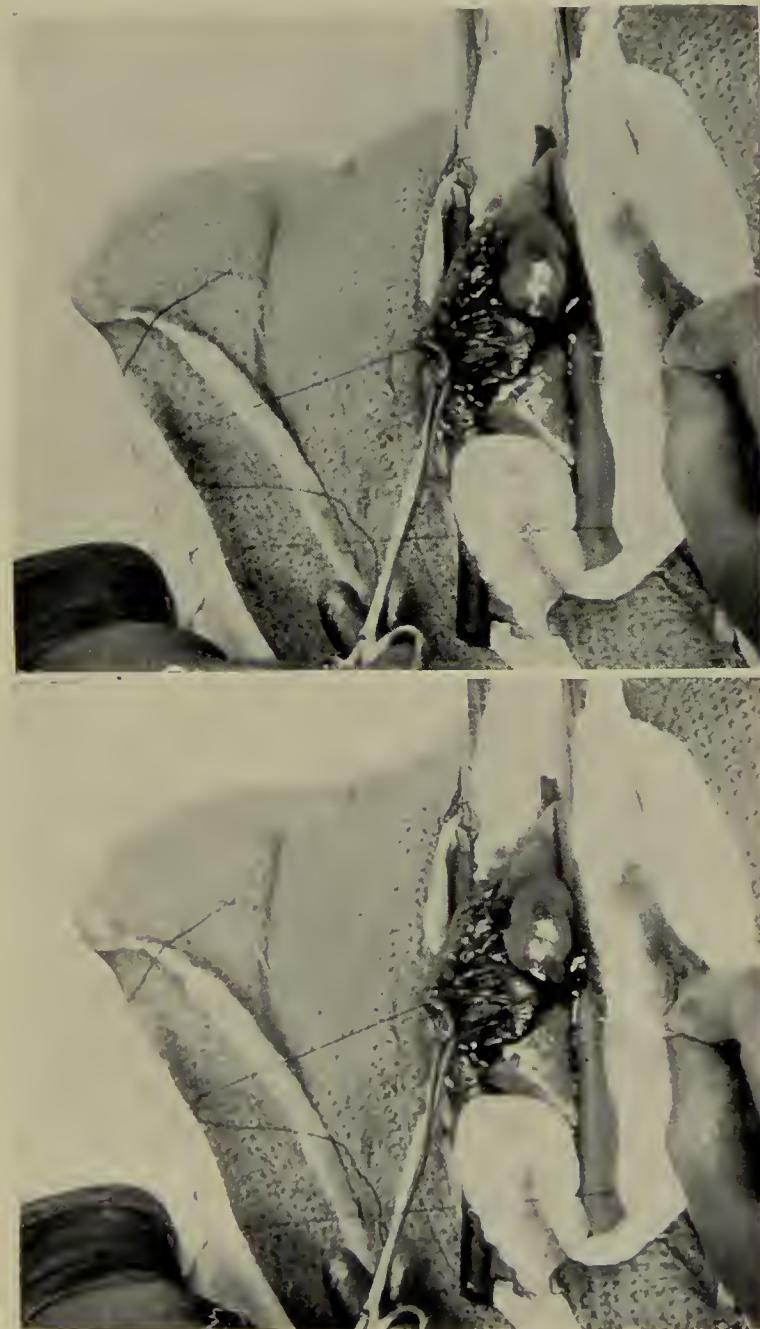
FIG. 14.—The posterior layer of hæmostatic suture nearly completed.

PLATE XII.—ANTERIOR GASTRO-JEJUNOSTOMY.
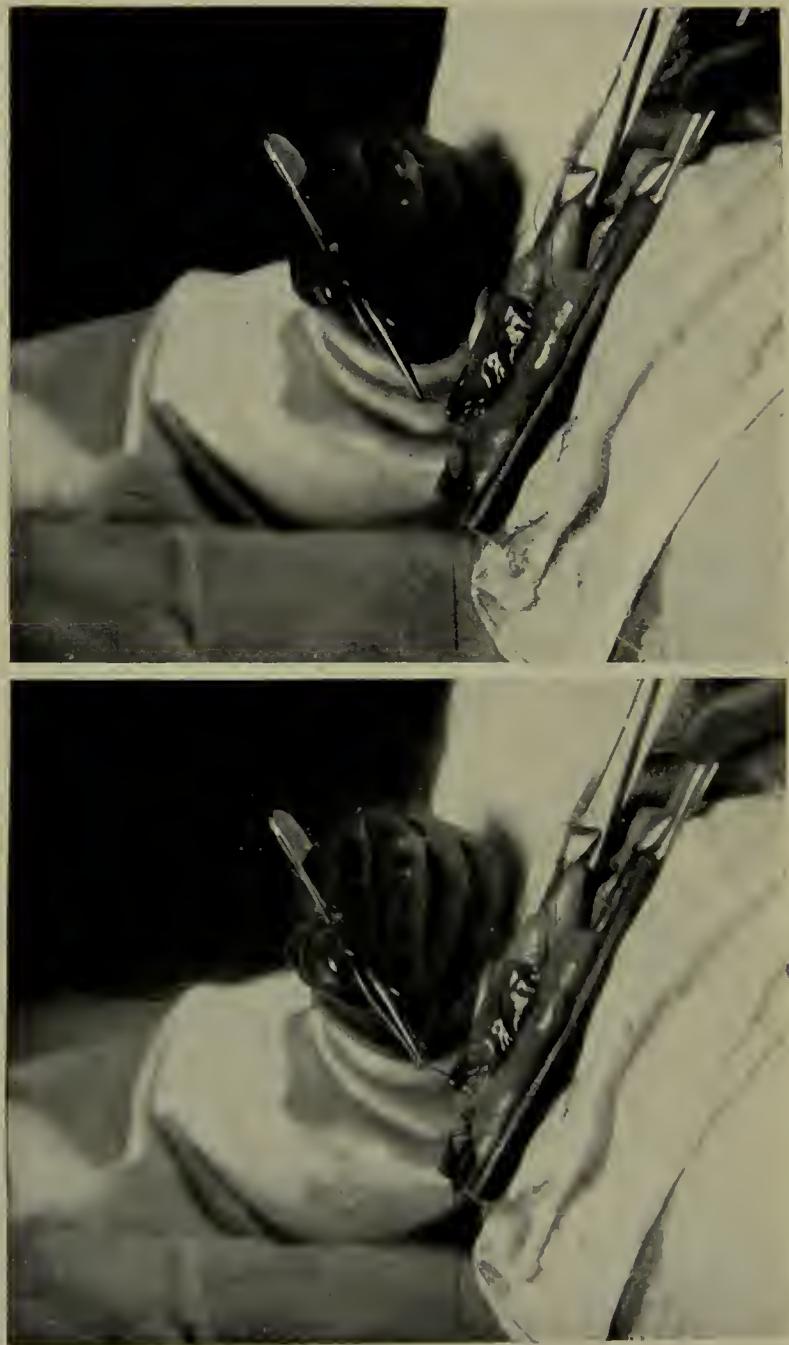
FIG. 15.—The last stitch of the posterior layer. Note the needle emerging from the mucous surface of the stomach.

On reaching the right extremity of the opening it is necessary to reverse the direction of the suture. This is accomplished as follows : After passing the stitch at the extreme right end of the opening, the needle emerges on the mucous surface of the stomach (Plate XII, Fig. 15).

Then the needle is passed again through the stomach wall from the mucous surface (Plate XIII, Fig. 16),

PLATE XIII.—ANTERIOR GASTRO-JEJUNOSTOMY.

Fig. 16.—Reversal of direction of stitch. The needle is being passed from the mucous surface back again through the stomach wall.

PLATE XIV.—ANTERIOR GASTRO-JEJUNOSTOMY.
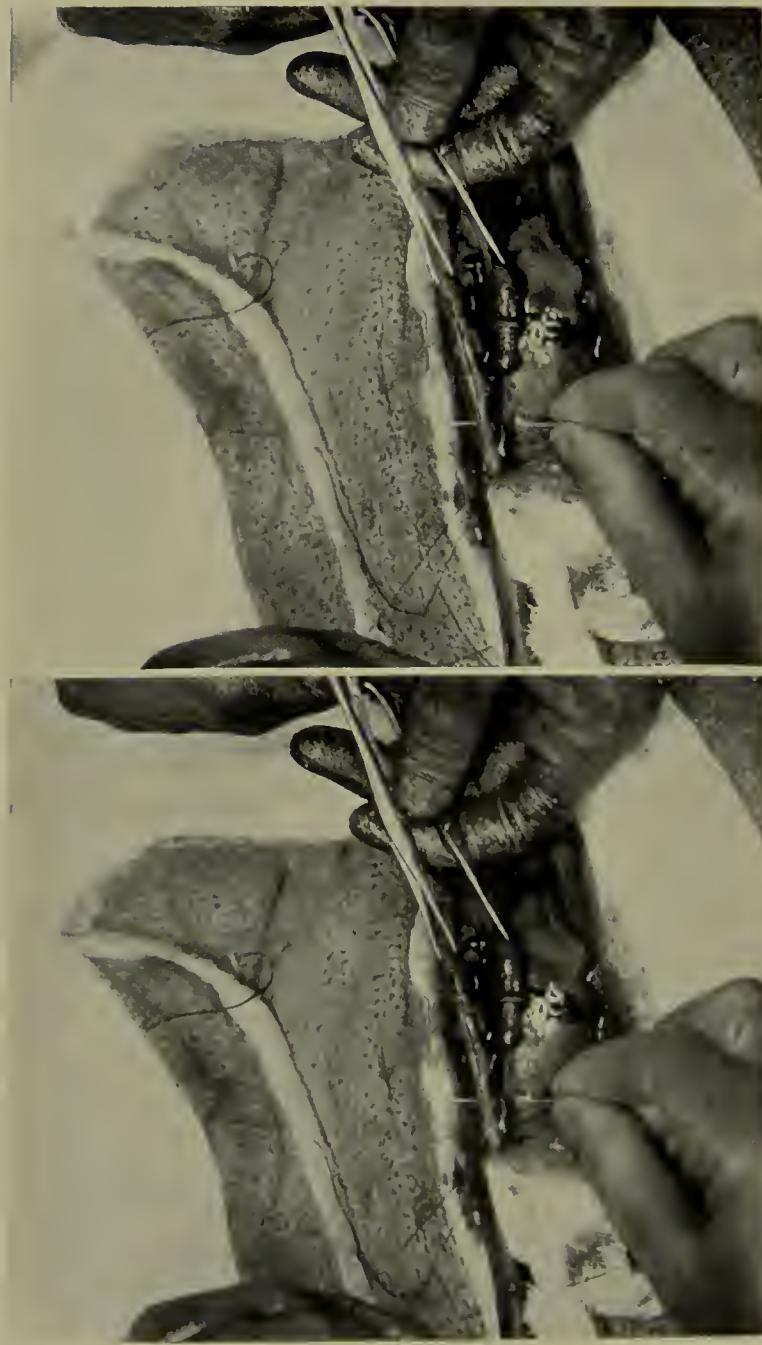
FIG. 17.—The first stitch of the anterior layer. Note that the needle is being passed from the peritoneal surface of jejunum and emerges on peritoneal surface of stomach.

and then the suture is continued by passing the needle through the jejunum from the peritoneal aspect, then through the mucous and muscular coats of the stomach to the peritoneal surface (Plate XIV, Fig. 17).

While the anterior layer of the hæmostatic suture is being passed the walls of the stomach and jejunum are held up by means of two pairs of Porrier's forceps, which are applied at the left extremity of the opening and held by the assistant.

In this way the mucosa is everted and any bleeding vessels can be seen readily when the clamps are removed.

The careful application of this suture is of great importance for two reasons : first, if the stitches be inserted at too great intervals, or be not pulled sufficiently taut, fatal hæmorrhage may ensue from the mucous membrane ; secondly, if the sutures be pulled too tightly, or the mucosa of the jejunum be not in accurate apposition with the gastric mucosa, ulceration will occur along the line of suture (gastro-jejunal ulcer).

The stitches should be at intervals of not more than one-eighth of an inch.

PLATE XVI.—ANTERIOR GASTRO-JEJUNOSTOMY.

FIG. 19.—The serous suture completed. The edges of the gastric and jejunal mucous membranes are now invisible.

The two ends are then drawn taut and cut short (Plate XVI, Fig. 19), the anastomosis gently washed with warm saline solution, and all pads removed. By the completion of the serous suture, the mucous membranes of the stomach and jejunum are buried, and so are visible no longer.

The next step is a most important part of the operation, namely, the application of " anchor " sutures to prevent kinking of the gut, or the formation of a spur.   I insert two or three sutures between the stomach and jejunum at each end of the anastomosis (Plates XVII and XVIII, Figs. 20 and 21).   In this way the abrupt angles which the afferent and efferent limbs of the jejunum make at each end of the anastomosis are changed into a gradual curve, and the patency of the opening between the stomach and efferent loop of the jejunum is assured.

The anastomosed viscera are then returned into the abdomen, and the abdominal wound sewn up as usual.

The operation should be completed in twenty-five or thirty minutes.   The points of importance in the operation are :

1. Making a large anastomotic opening, at least three inches in length.

2. Excision of a portion of the gastric mucosa.

3. Careful passing of the hæmostatic suture to secure firm and accurate apposition of the gastric and jejunal mucous membrane.

4. The use of " anchor " sutures.

PLATE XVII.—ANTERIOR GASTRO-JEJUNOSTOMY.
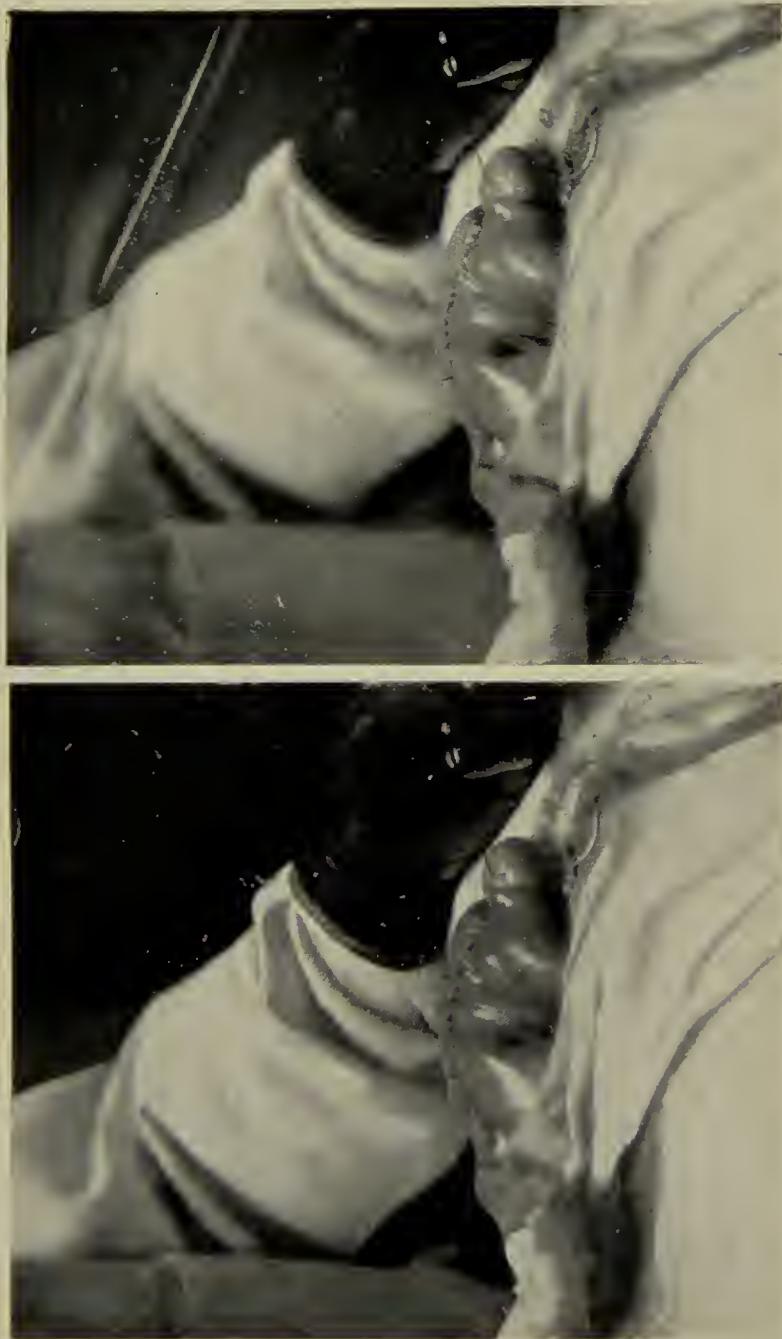
FIG. 20.—The insertion of two "anchor sutures" at left extremity of anastomosis.

PLATE XVIII.—ANTERIOR GASTRO-JEJUNOSTOMY.

FIG. 21.—The insertion of two "anchor sutures" at the right extremity of anastomosis.

Posterior Gastro-jejunostomy.—The method of gastro-jejunostomy in general use is spoken of as the posterior " no-loop " operation. This description is not strictly accurate. It is impracticable to make the anastomosis absolutely close to the duodeno-jejunal junction. In practice
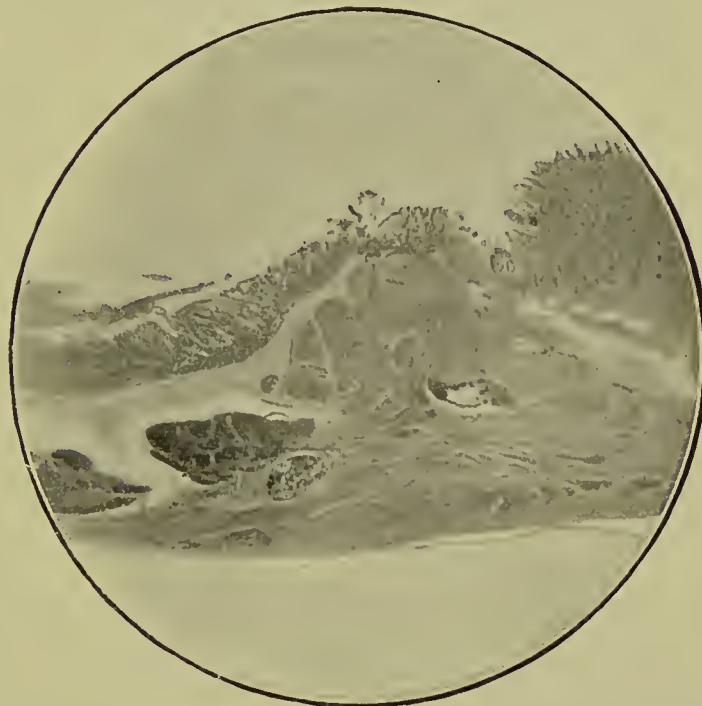
Fig. 22.—Section of the junction between the stomach and jejunum five months after Gastro-jejunostomy.

The continuity of the mucous membrane is well shown.

about an inch of jejunum intervenes between the origin of the jejunum and the point at which the junction is made. It is more correct, therefore, to speak of the posterior " short-loop " operation. It is performed in the following manner :

After opening the peritoneal cavity, the stomach and transverse colon are drawn gently upwards, and two fingers introduced into the abdomen seek the origin of the jejunum close to the left of the spine at the root of the transverse meso-colon.

The posterior surface of the stomach is exposed in the
following manner : A bloodless portion of the meso-colon,
close to the root of the jejunum, is seized with a pair of
forceps. The forceps are lifted up and a nick made by the
side of them with a pair of scissors. The opening is en-
larged by stretching until it is about four inches in length.

In Fig. 23, Plate XIX, the posterior wall of the stomach
is exposed, the edges of the meso-colon are held in forceps,
and the origin of the jejunum is grasped by the thumb and
forefinger of the right hand.

PLATE XIX.—POSTERIOR GASTRO-JEJUNOSTOMY.
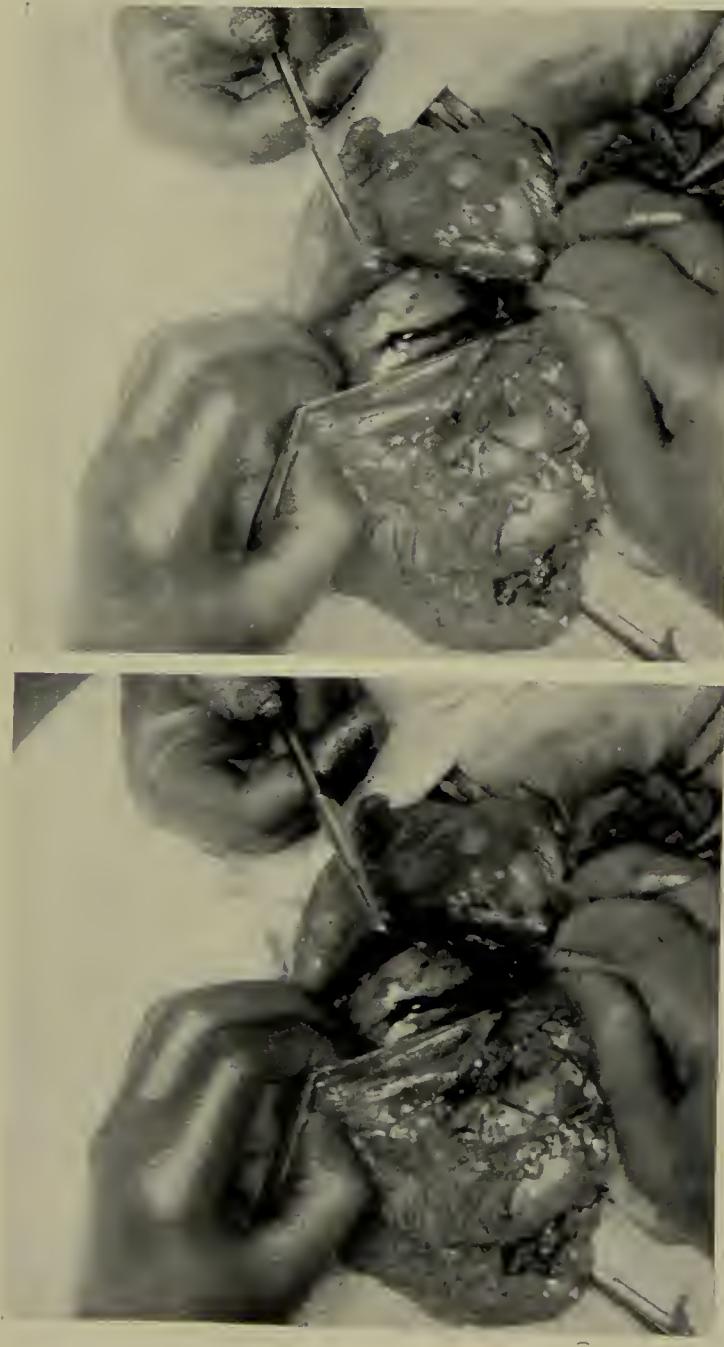
FIG. 23.—Posterior surface of stomach exposed ; the origin of jejunum is shown grasped by the thumb and forefinger of right hand.

Plate XX.—POSTERIOR GASTRO-JEJUNOSTOMY.

Fig. 24.—The jejunum is held by the operator while the assistant applies the clamps.

A portion of jejunum four inches long, close to its origin, is clamped with a pair of long forceps, the blades of which are sheathed with rubber (Plate XX, Fig. 24). The jejunum is clamped so that its direction is from right to left—that is, so that the portion nearest its origin is at the right end of the clamp.

A portion of the posterior wall of the stomach is drawn up through the hole which has been made in the transverse meso-colon, and is clamped with a second pair of long forceps. The portion clamped should be at least four inches long, and should be as near the pyloric end of the stomach as is possible. The clamp is applied to the stomach obliquely from above downwards and to the right. [Thus when the anastomosis has been completed and the stomach returned to the abdomen, the direction of the anastomotic opening will be oblique, extending from above downwards and to the left. The anastomosis is therefore anti-peristaltic.]

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The transverse colon is now returned into the abdomen, the handles of the forceps are taken charge of by the assistant, and the viscera to be anastomosed are surrounded with pads. Nothing is visible except the portions of stomach and jejunum to be anastomosed (Plate XXI, Fig. 25).

PLATE XXI.—POSTERIOR GASTRO-JEJUNOSTOMY.
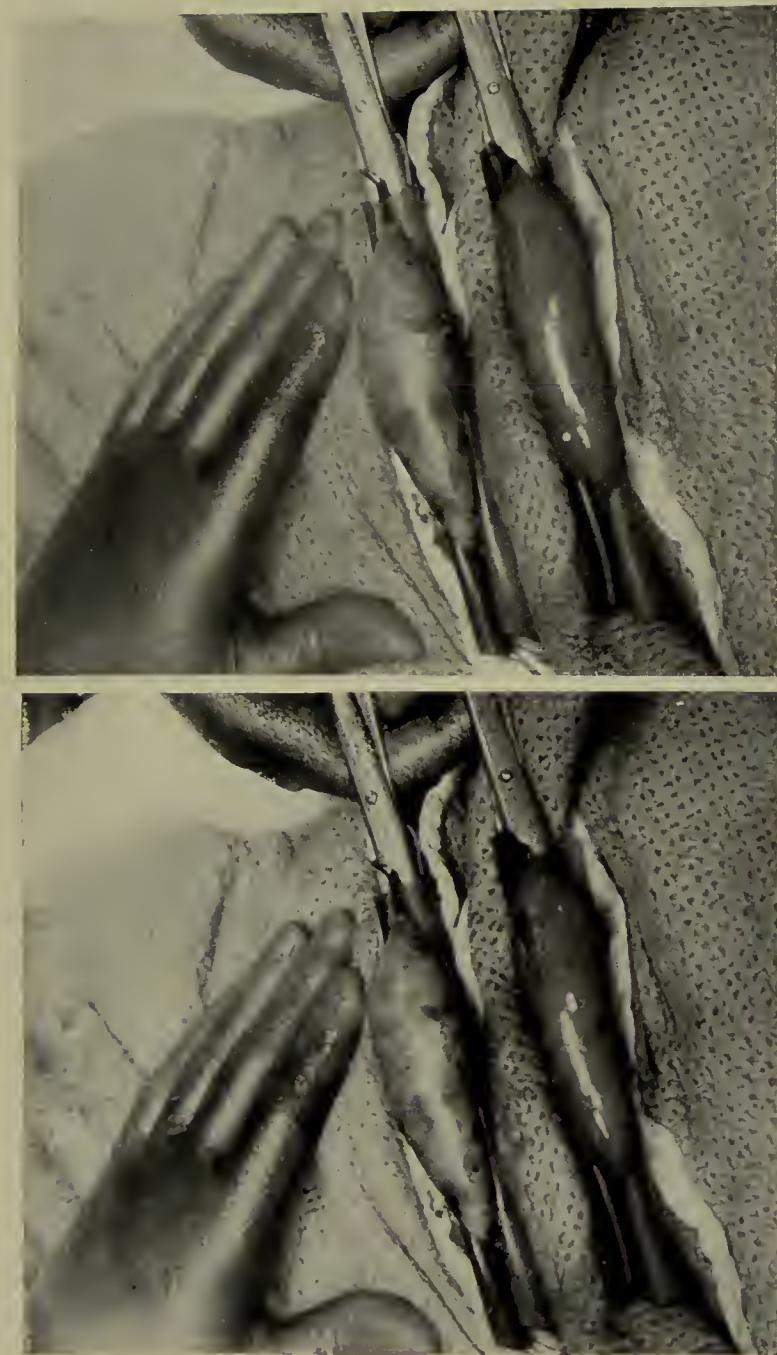
FIG. 25.—Portions of stomach and jejunum clamped and surrounded by pads.

PLATE XXII.—POSTERIOR GASTRO-JEJUNOSTOMY.
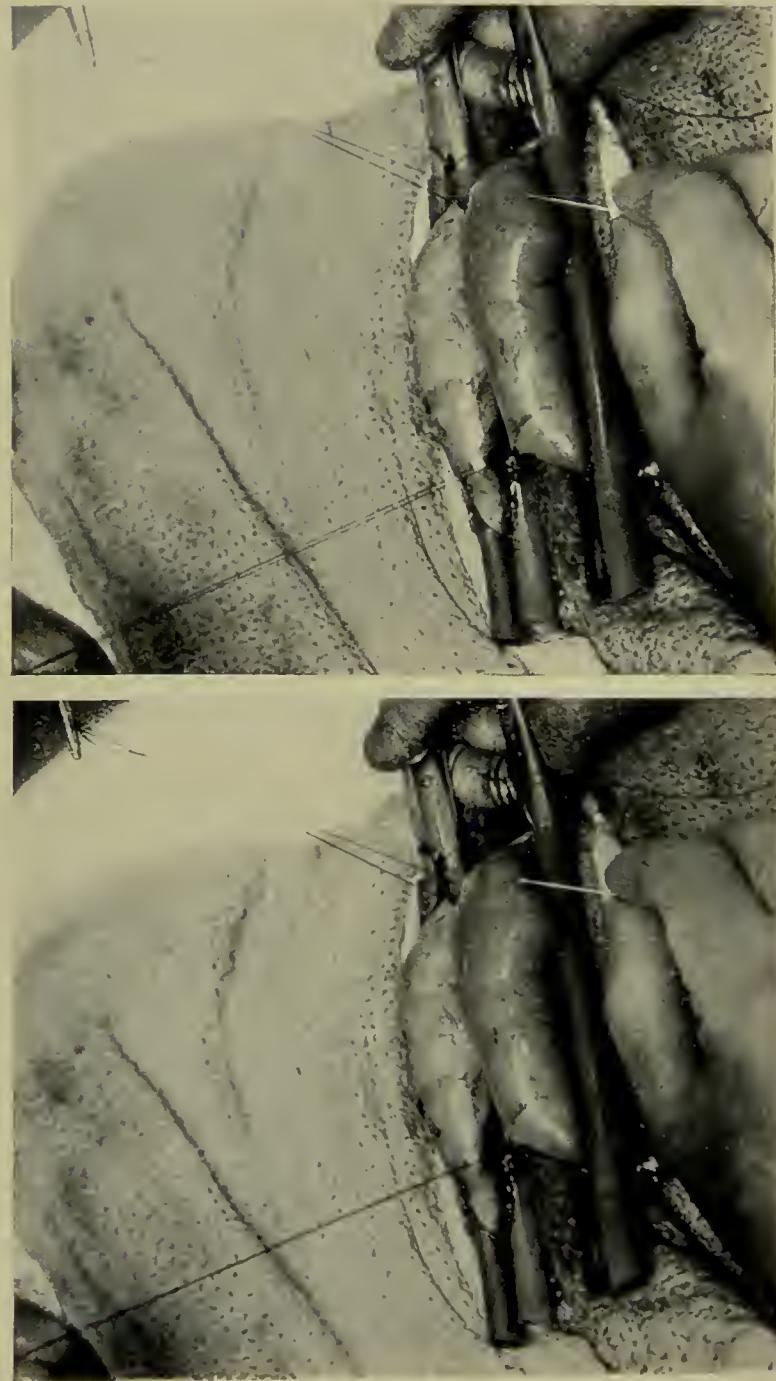
FIG. 26.—The clamped portions of viscera united at their extremities by the insertion of two guide sutures.

A guide suture is inserted at each end of the clamped viscera (Plate XXII, Fig. 26). These sutures are at least four inches apart, and their object is to ensure that the line of suturing shall be straight.

The anastomosis is now effected by means of two continuous sutures, an inner hæmostatic suture penetrating all the coats of the viscera, and an outer approximating suture including the peritoneal, muscular, and submucous coats. For both sutures I use a very fine black Irish linen thread.

The outer serous suture is commenced at the left end of the clamped portions of the stomach and jejunum, and is continued towards the right for four inches (Plate XXIII, Fig. 27).

PLATE XXIII.—POSTERIOR GASTRO-JEJUNOSTOMY.
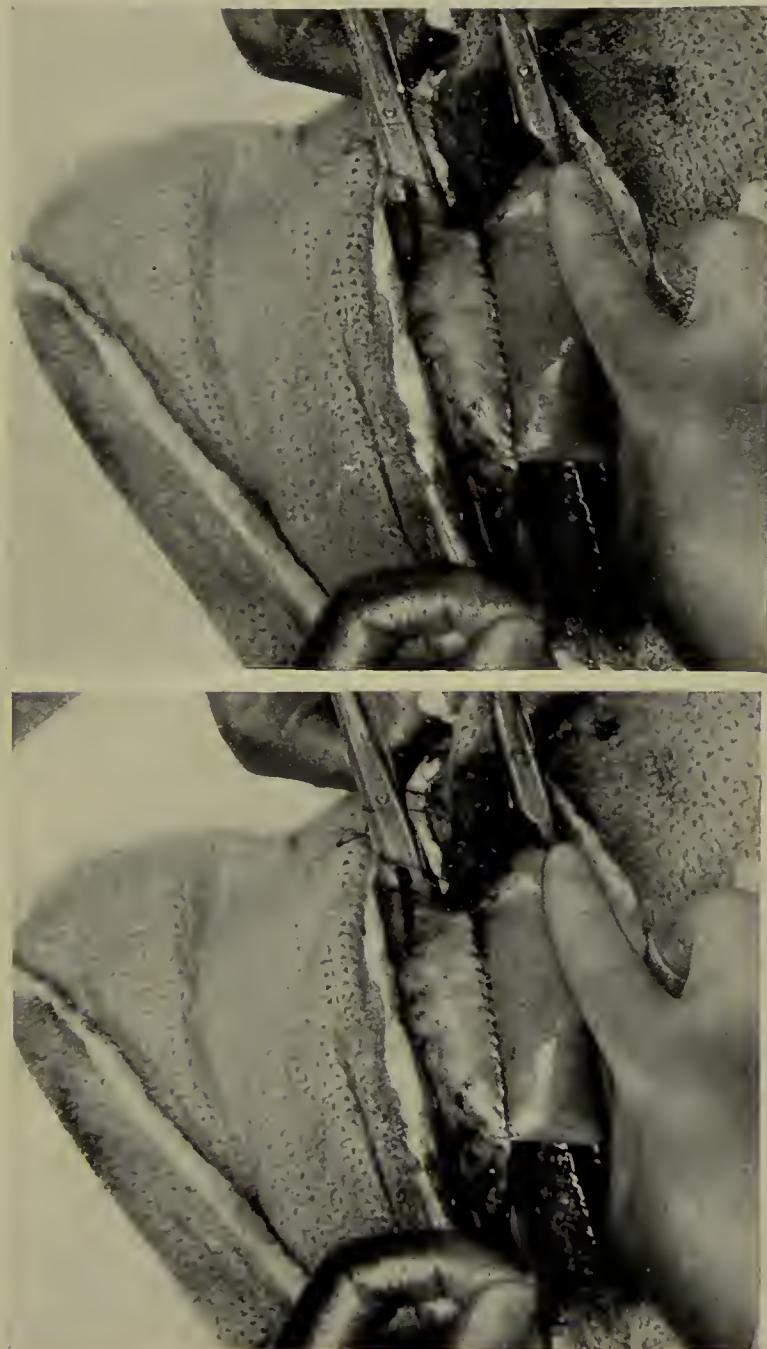
FIG. 27.—The posterior half of the serous suture complete.

PLATE XXIV.—POSTERIOR GASTRO-JEJUNOSTOMY.
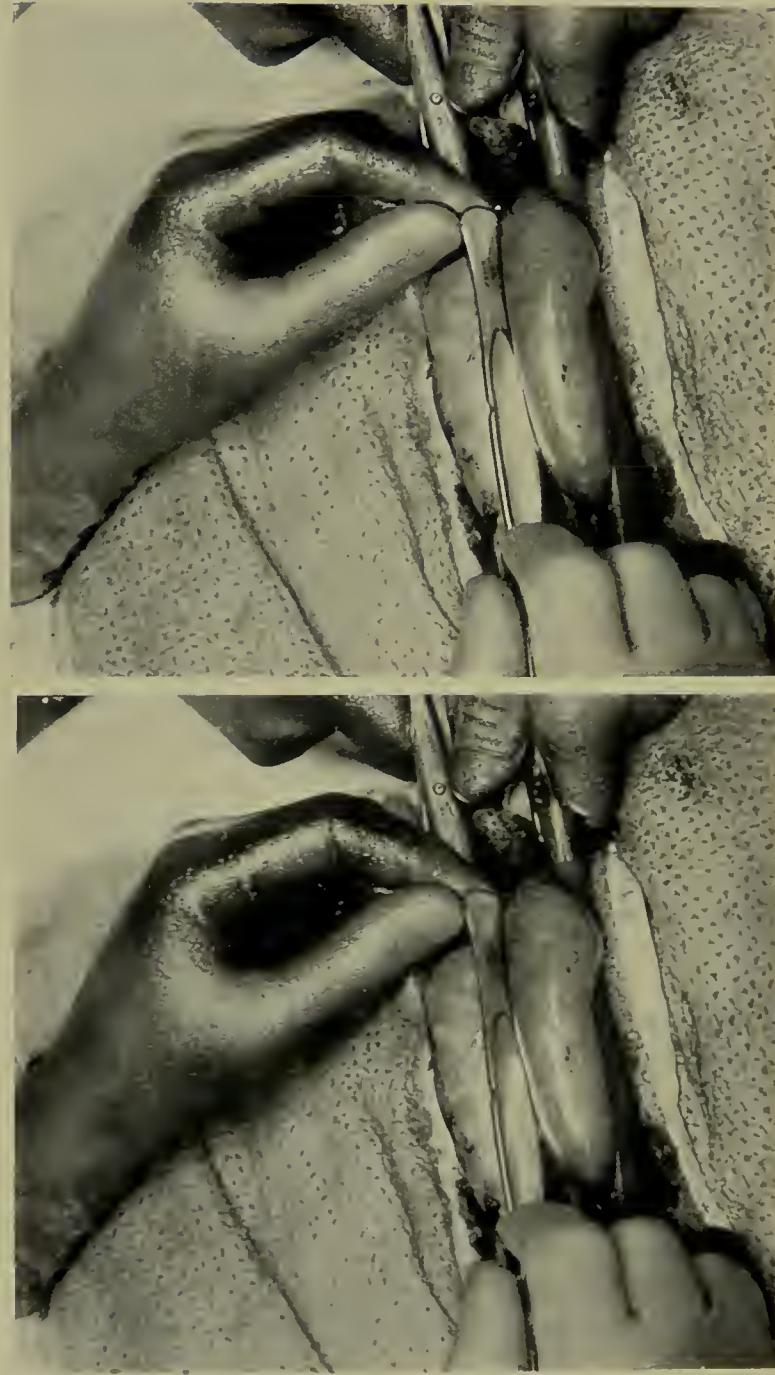
FIG. 28.—Measuring, to make sure that the portion sutured measures four inches.

I have a notch cut on the handles of my scalpels $2\frac{1}{2}$ inches from the end, so that by using this as a measure (Plate XXIV, Fig. 28), I can be certain that the line of suture is of adequate length. Without being unthreaded, the needle is clamped with a pair of pressure forceps and laid upon the muslin sheet above the wound, ready for use later. I find my " grip-eyed " needles of great advantage, as they cannot become unthreaded accidentally.

A portion of the stomach wall, as close to the suture line as possible, is picked up with a pair of dissecting forceps, and the stomach is opened by cutting into it with a small pair of scissors (Plate XXV, Fig. 29). The incision is then prolonged in both directions until the opening measures three inches. A similar opening is made in the jejunum. I attach great importance to making the anastomotic opening of sufficient size.

As the viscera are opened the mucous membrane pouts into the wound. With a pair of scissors, about a quarter of an inch of the mucous membrane is removed on each side of the opening into the stomach. As a rule I do not remove any of the jejunal mucosa.

PLATE XXV.—POSTERIOR GASTRO-JEJUNOSTOMY.
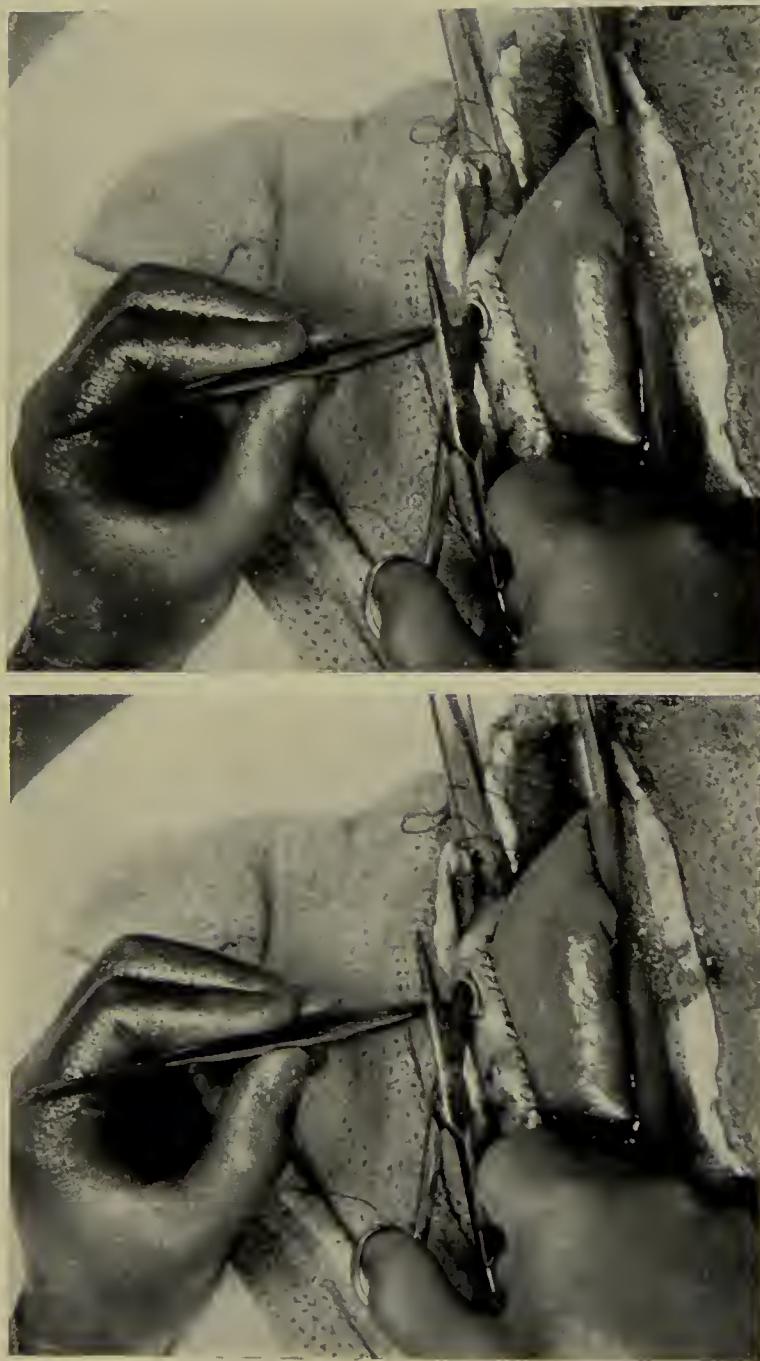
FIG. 29.—Opening the stomach.

Plate XXVI.—POSTERIOR GASTRO-JEJUNOSTOMY.
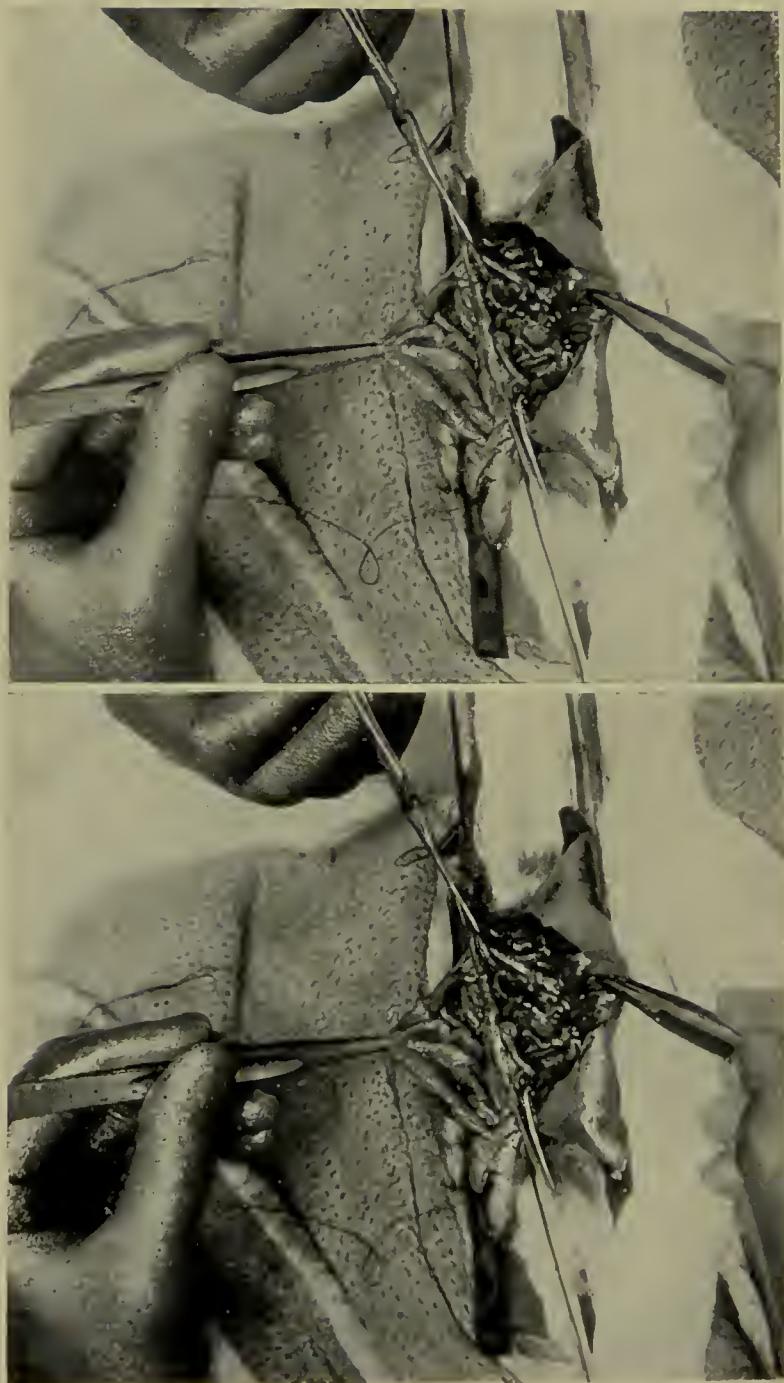
Fig. 30.—Stomach and jejunum opened. The walls of the stomach and jejunum held in apposition by two pairs of Porrier's forceps, ready for insertion of posterior layer of hæmostatic suture.

The inner hæmostatic suture, penetrating all coats of the stomach and jejunum, is now introduced.   The posterior cut edges of the walls of the stomach and jejunum are kept in apposition by means of two pairs of Porrier's forceps, one pair being applied at each end of the anastomotic opening (Plate XXVI, Fig. 30).

The inner suture is commenced at the same point as the serous suture, and is continued (Plate XXVII, Fig. 31) without interruption to the starting-point.

In the insertion of the posterior layer the needle is passed through the mucosa of the jejunum, then through the wall of the jejunum, the wall of the stomach, and finally through the mucosa of the stomach ; but on reaching the right extremity of the opening it is necessary to reverse the direction of the suture.

Plate XXVII.—POSTERIOR GASTRO-JEJUNOSTOMY.

Fig. 31.—The posterior layer of the hæmostatic suture nearly completed.

PLATE XXVIII.—POSTERIOR GASTRO-JEJUNOSTOMY.

FIG. 32.—The last stitch of the posterior layer. Note the needle emerging from the mucous surface of the stomach.

This is accomplished as follows: After passing the stitch at the extreme right of the opening the needle emerges on the mucous surface of the stomach (Plate XXVIII, Fig. 32).

Then the needle is passed back again through the stomach
wall from the mucous surface (Plate XXIX, Fig. 33),

PLATE XXIX.—POSTERIOR GASTRO-JEJUNOSTOMY.

FIG. 33.—Reversal of direction of stitch. The needle is being passed from the mucous surface back again through the stomach wall.

PLATE XXX.—POSTERIOR GASTRO-JEJUNOSTOMY.
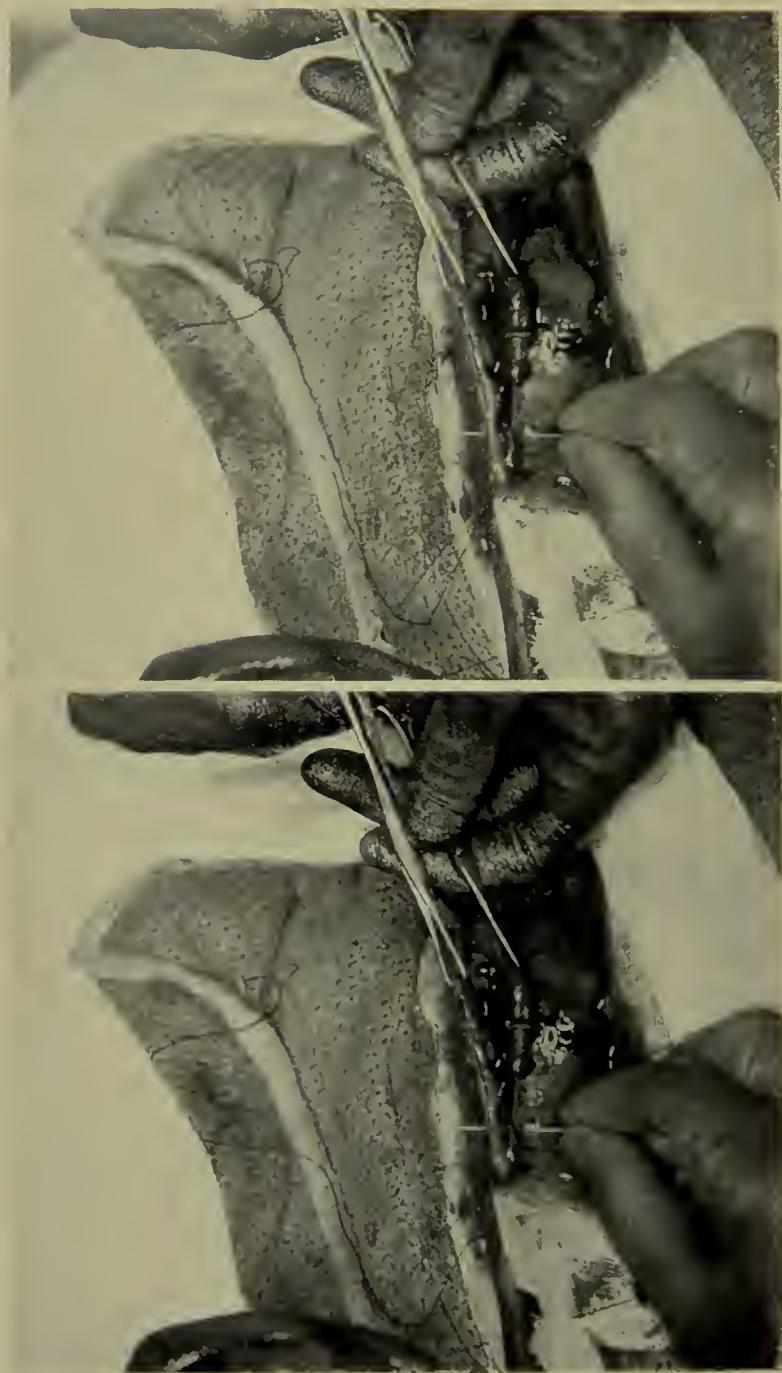
FIG. 34.—The first stitch of the anterior layer. Note that the needle is being passed from the peritoneal surface of jejunum and emerges on peritoneal surface of stomach.

and then the suture is continued by passing the needle through the jejunum from the peritoneal aspect, then through the mucous and muscular coats of the stomach to the peritoneal surface (Plate XXX, Fig. 34). The passing of the suture is facilitated by holding up the left extremities of the incisions into the stomach and jejunum by means of two pairs of Porrier's forceps. By passing the suture in this manner the edges of the mucosa are everted, and any bleeding vessels can be seen readily when the clamps are removed. The careful application of this suture is of great importance because, first, if the stitches be not inserted sufficiently closely together, or be not pulled sufficiently taut, fatal hæmorrhage may ensue from the mucous membrane ; secondly, if the sutures be pulled too tightly, or the mucosa of the jejunum be not in accurate apposition with the gastric mucous membrane, ulceration will occur along the line of suture (gastro-jejunal ulcer). The stitches should be at intervals of not more than one-eighth of an inch.

On reaching the starting-point the two ends of the suture are tied, the knot being on the mucous surface, and the ends cut short (Plate XXXI, Fig. 35). The clamps, having served their purpose, are now taken off.

If there be any bleeding point on the mucosa, it is controlled by passing a through-and-through suture.

PLATE XXXI.—POSTERIOR GASTRO-JEJUNOSTOMY.
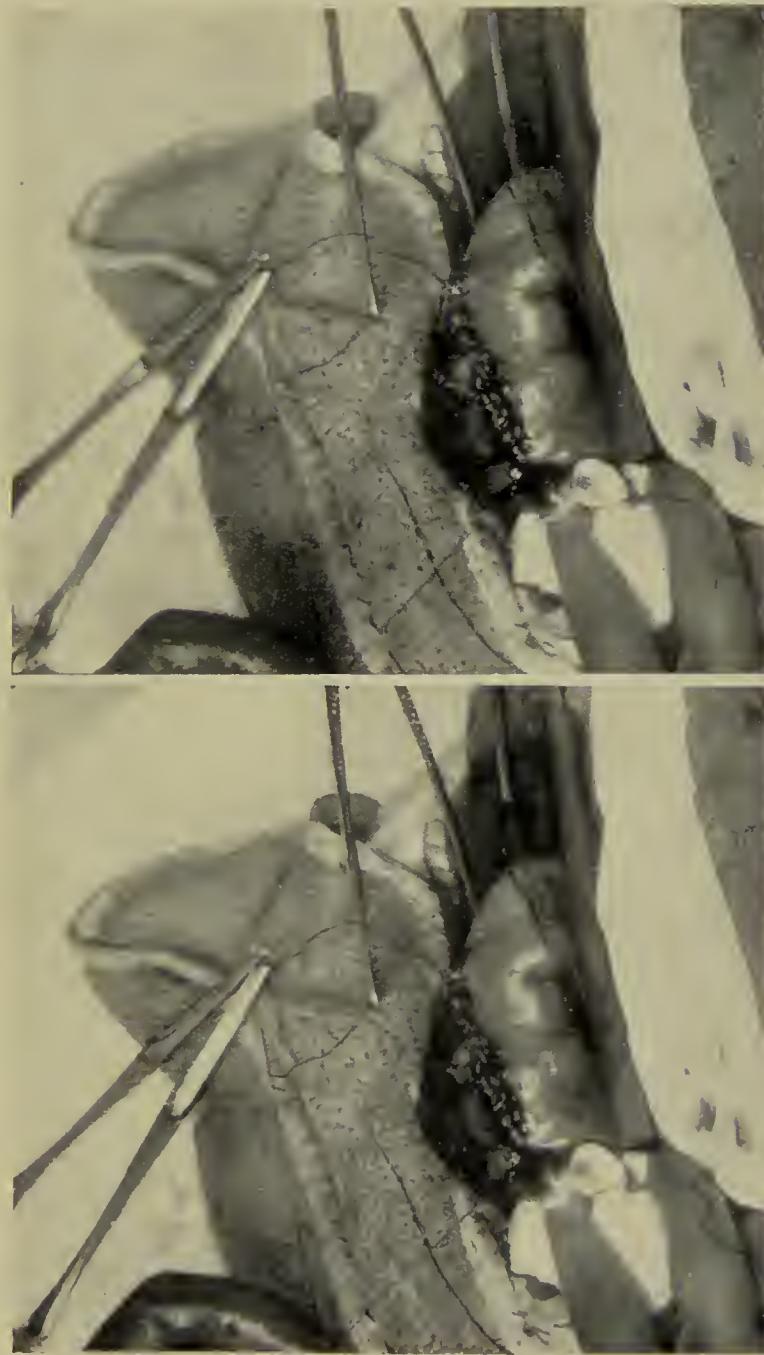
FIG. 35.—The hæmostatic suture is completed. Note that the cut edges of the gastric and jejunal mucous membranes are everted and so are still visible.
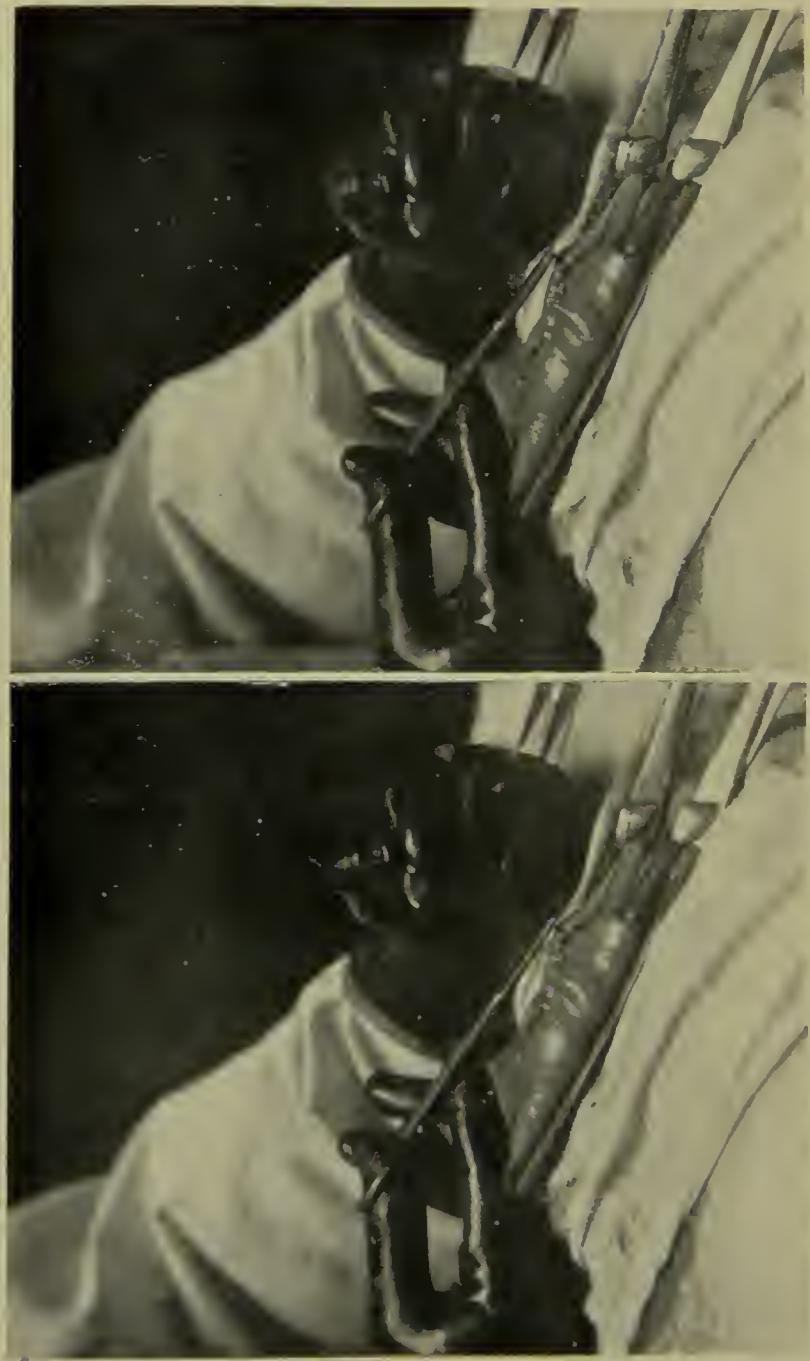
FIG. 36.—The anastomosis completed. The mucous membranes have been buried by the completion of the serous suture.

The outer serous suture is now resumed, and is continued round the right extremity of the anastomotic opening and then in front of the inner suture to the original starting-point.

The two ends are drawn taut and cut short (Plate XXXII, Fig. 36), the anastomosis gently washed with warm saline solution, and all pads are removed.

The completion of the serous suture buries the everted mucous membranes, which are visible no longer.

The edges of the hole in the meso-colon are now fixed to the stomach round the anastomosis by means of four sutures, so that no gap is left to allow prolapse of the intestine through the hole in the meso-colon and so lead to an internal hernia.

The abdominal wound is sewn up in the usual way.

Roux's Gastro-jejunostomy "En-Y."—In this method first suggested by Wölfler and improved and popularised by Professor Roux, the jejunum is cut across nine or ten inches from its origin, and the distal end anastomosed to the posterior surface of the stomach through an aperture made for the purpose in the transverse meso-colon. Then the proximal end of the jejunum is implanted into the side of the distal portion three inches below its attachment to the stomach.

The anastomosis to the stomach may be made equally well on the anterior surface of the viscus.

The advantage claimed for Roux's operation is that it prevents regurgitant vomiting. This is not absolutely true,

as I have known at least two cases in which regurgitant vomiting was present after this method had been employed.

It is a complicated and difficult operation, and takes at least from fifteen to twenty minutes longer to perform than a simple gastro-jejunostomy. The chief, and to my mind fatal objection to it is, that the danger of jejunal ulcer is increased, as a portion of jejunum is exposed to the action
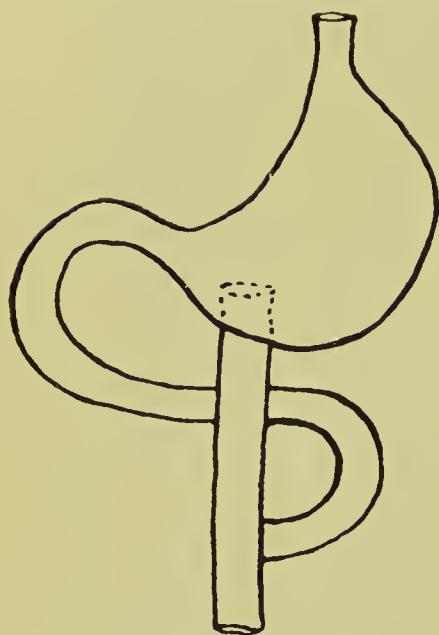
Fig. 37.—Gastro-jejunostomy " En-Y "   Fig. 38.—Gastro-jejunostomy " En-Y "
(Roux's method).   (Moynihan's modification).

of the gastric juice, without the neutralising effects of the bile and pancreatic juice.

The method is not one to be recommended as the operation of choice, but in the treatment of jejunal ulcer, and regurgitant vomiting, it is sometimes the only procedure available. I have performed it for these conditions on several occasions. The results are satisfactory on the whole, but it has seemed to me that the opening from the stomach is not sufficiently large, and so a certain degree of stasis is present after the operation.

This objection can be overcome by anastomosing the

jejunum to the stomach laterally, as suggested by Sir Berkeley Moynihan, instead of in the manner originally carried out by Professor Roux.

I consider Sir Berkeley's modification to be preferable to the older method. The operation is performed as follows : A portion of the stomach is clamped as for the ordinary anterior operation. Two clamps are then placed on the jejunum eight or nine inches from its origin, and the intestine divided between the clamps, the cut being extended into the mesentery. The distal end of the jejunum is now closed by two continuous sutures, an inner, penetrating all coats, and an outer serous suture.

This portion of the jejunum is anastomosed to the stomach exactly as in an anterior gastro-jejunostomy. Finally, the proximal end of the jejunum is implanted into the left side of the distal portion of the jejunum, three inches below its attachment to the stomach.

6. GASTRO-PLASTY AND GASTRO-GASTROSTOMY

Gastro-plasty is the application to the stomach of the principle of the operation of pyloroplasty. Its rôle is a very limited one. It has been performed frequently for hour-glass stomach, but the after-results are not satisfactory. Relapse ensues in 25 per cent. of the cases. To guard against relapse after gastro-plasty, gastro-jejunostomy should be performed in addition. If, however, a gastro-jejunostomy be performed, the gastro-plasty in most cases is un-necessary.

Gastro-plasty is performed as follows : After opening the abdomen the stomach is packed round with pads to prevent contamination of the peritoneum. An incision $3\frac{1}{2}$ to 4 inches is made in the long axis of the stomach, having its centre at the constriction. The edges of the stomach at the middle of the incision are seized with forceps. These are gently

pulled apart, so as to change the direction of the incision from the long axis to the vertical axis of the stomach. The forceps are held by an assistant, and the incision is sewn up with two continuous sutures of linen thread, an inner suture penetrating all coats, and an outer serous suture. The abdominal wound is closed in the usual manner without drainage.

Gastro-gastrostomy.—In gastro-gastrostomy the pouches on each side of the constriction are anastomosed, so as to make a free passage from the cardiac to the pyloric pouch below the contracted isthmus.

The operation is performed in the following manner: The stomach is clamped on either side of the constriction. A continuous serous suture is then passed from the constriction to the greater curvature, so as to bring into apposition the cardiac and pyloric pouches below the constricted area. The needle is then laid aside, and incisions are made into each pouch in front of the suture line, so as to open the interior of both pouches. All redundant mucosa is excised, and a continuous suture of linen, penetrating all coats, is passed right round the opening. Then the original serous suture is continued in front of the anastomosis. The technique is exactly the same as that of gastro-jejunostomy except that stomach is united to stomach, instead of jejunum to stomach.

Gastro-gastrostomy is open to the same objections as gastro-plasty, namely the frequency with which relapse ensues. In at least 30 per cent. of the cases the patient does not obtain permanent relief.

7. GASTRECTOMY

Gastrectomy may be partial or complete.

(a) **Partial Gastrectomy.**—In partial gastrectomy for cancer, at least one inch of the duodenum, one-half of the

greater curvature, and the whole of the lesser curvature should be removed, together with the glands lying along the lesser curvature, and those connected with the pyloric half of the greater curvature.

Personally, I prefer to make the section of the greater curvature at the junction of the middle third with the cardiac third, so that two-thirds of the stomach are removed.

The steps of the operation are as follows :

(i) An opening is made through the gastro-hepatic
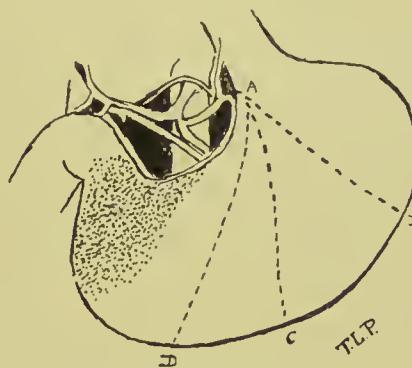
FIG. 39.—Lines of incision used in partial gastrectomy.

A B—Line of incision advisable except in very early cases (Mayo, Moynihan, the author).
A C—Line of incision in very early cases (Mikulicz).
A D—Line of incision of Hartmann.

omentum so as to open the lesser peritoneal sac. The limits of the growth are explored thoroughly, so as to ascertain whether the case is suitable for excision. Adhesion to the pancreas is not necessarily a contra-indication to resection if the conditions be favourable otherwise. According to Haberkant, removal of a portion of the pancreas raises the mortality rate to 76 per cent., and Mikulicz gives 74 per cent. as the death-rate in such cases. Dr. W. J. Mayo does not consider that there is any special mortality due to operable pancreatic complication.

In one of my cases, there was a freely movable, large mass attached to the lesser curvature and pylorus, which I took to be infiltrated omentum. When proceeding to cut

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away the stomach after the ligation of the vessels and the dissection had been completed, I found to my dismay that the mass consisted of the head of the pancreas, which had been lifted out of its bed by the pyloric growth. There was no alternative but to remove about two-thirds of the head of the pancreas with the stomach. This I did, and the patient made an excellent recovery, complicated only by a pancreatic fistula which persisted for three weeks. The operation was five years ago, and the patient has been in excellent health ever since.

Of greater importance is the extent of glandular infection. Enlargement of glands does not necessarily mean that they are infected with carcinoma. This cannot be determined except by microscopical examination. If, therefore, there be enlarged glands which cannot be removed completely, one of them should be excised and submitted to microscopical examination, the resection postponed until a later date, and the gastro-jejunostomy performed meanwhile.

(ii) Having decided that the case is one suitable for resection, the next step is to make the anastomosis between the jejunum and the stomach. Most operators leave this step until the last. In my Hunterian lectures I suggested that the gastro-jejunostomy should be performed before the resection. My reasons for this are as follows: First if the gastro-jejunostomy be left to the last there is a risk that the surgeon may be tempted to perform it hurriedly owing to the condition of the patient; secondly, after the gastro-jejunostomy has been completed, the surgeon is in a better position to judge how the patient will stand the additional operation of resection; thirdly, the gastro-jejunostomy is performed more easily before resection; and fourthly, in the case of a tumour of doubtful nature, or when enlarged, inoperable glands are present, the time occupied can be utilised to freeze, and to examine microscopically, a portion of

the tumour or of the suspicious gland. Such investigation, however, requires a pathologist specially skilled in such work, and the report is of most value if it negative malignant disease. I do not act on a positive report of cancer made under these conditions, unless I feel sure from the clinical aspect of the case that the growth is malignant.

The only objection to performing the gastro-jejunostomy first is, that the site chosen for the anastomosis may be in the way of an adequate resection. This objection is met by making the anastomosis as far to the left as is possible.

Whether an anterior or posterior gastro-jejunostomy be performed is immaterial. If the resection is to be a wide one, usually it is advisable to make the gastro-jejunostomy on the anterior wall of the stomach. Whichever method be selected, it is performed in exactly the same way as described previously.

The surgeon now decides whether he will complete the operation by performing resection, or whether he will postpone the remainder of the operation until a later date. There is much to be said for the operation *à deux temps*, as suggested originally by Tupolske. The chief argument in favour of it is, that by delaying the resection the patient can be fed up, and so be in a better condition to undergo the severer operation. I think, however, this depends on the type of the growth. Those patients who have a hard growth at the pylorus, which has caused partial obstruction, and so resulted in great emaciation, undoubtedly will be benefited by the gastro-jejunostomy, and will put on weight rapidly. Those patients, on the other hand, who have a soft ulcerating or fungating growth, and are suffering from toxæmia rather than from obstruction, will not improve to any extent, and so delay will not improve necessarily their prospects of recovery.

Then again, if the resection be postponed, recent adhesions,

due to the first operation, are often troublesome, and may make what would have been, in the first instance, an easy resection, a very difficult one. Another objection to postponing the resection is that sometimes the patient improves so rapidly that the second operation is declined. To a sensible patient, however, the position can be explained beforehand. If, at the completion of the gastro-jejunostomy, the patient's condition be fairly satisfactory, I think it is better to complete the operation.

(iii) If the surgeon decide to perform partial gastrectomy, the next step is the ligation of the four blood-vessels, the gastric, the superior pyloric, the gastro-duodenal, and the left gastro-epiploic arteries.

The stomach is drawn well downwards and to the right, and the gastric artery is felt for with the left forefinger. A curved needle armed with a double thread of No. 2 catgut is now passed between the artery and the stomach as high up as possible. The artery is tied in two places, and divided below the ligature with a pair of scissors, the omentum being divided at the same time right up to the stomach.

(iv) The pyloric artery is sought for and tied with a double ligature, at a point as far away as possible from the duodenum, and divided between the two ligatures. From the point of ligation the gastro-hepatic omentum is divided with scissors, as close to the liver as possible, until the point of ligation of the gastric artery is reached.

(v) From the point of ligation of the gastric artery the glands and fat are dissected downwards along the lesser curvature for a distance of $1\frac{1}{2}$ inches. This is an important step in the operation, as to prevent recurrence it is essential to remove the whole of the lesser curvature and attached glands. Similarly, the fat and glands lying above the duodenum are dissected off the duodenum, from the right as far as the pylorus. By these dissections, the duodenum is

freed above, and a clear space is left at the left extremity of the lesser curvature.

(vi) Next, the gastro-colic omentum is dealt with. The left gastro-epiploic vessels are ligatured to the left of the proposed line of section. To the right of this point the gastro-colic omentum is dissected off the greater curvature for a distance of 1½ inches. The gastro-colic omentum is next ligatured in sections as close to the transverse colon as possible, so that all glands are left attached to the stomach. If the glands lying along the transverse colon be infected with cancer, the transverse colon ought to be resected, otherwise recurrence almost certainly will ensue (see p. 265).

In ligaturing the gastro-colic omentum great care must be taken not to wound the middle colic artery, as injury of this vessel probably will result in gangrene of a portion of the bowel. It has been stated by G. Feuerer, that if the middle colic artery be wounded, the danger of gangrene is obviated by wrapping the bowel round with omentum. Personally, I would hesitate to trust to this. It is better to take no risks. Wound of the middle colic artery indicates resection of the transverse colon. If resection of the transverse colon be necessary, it is simplest to perform a lateral anti-peristaltic anastomosis. This is easier than an iso-peristaltic one, as there is less difficulty in getting the parts in apposition. In some cases it may be necessary to divide the outer layer of the peritoneum at the splenic and hepatic flexures in order to bring together the two portions of bowel without tension.

(vii) The next step is the dissection of the sub-pyloric group of glands.

The omentum is separated upwards along the inferior border of the duodenum, the gastro-duodenal artery, lying in the groove between the duodenum and the pancreas behind

the pylorus, being ligatured. The omentum and the glands are raised upwards to the left, remaining attached to the pylorus and greater curvature of the stomach.

(viii) The posterior surface of the first two inches of the duodenum is separated from the pancreas, and a clean pad placed in position behind the duodenum.

(ix) A small pair of clamps is placed on the duodenum at the line of the proposed section, usually 1 to 1½ inches from the pylorus. A pair of pressure forceps is placed on the duodenum to the pyloric side of the clamp and the bowel divided between them. Both ends of the duodenum are cauterised with the actual cautery. The forceps holding the pyloric end of the duodenum is turned over to the right, and the cut surface of the duodenum is covered with a piece of gauze wrung out in warm saline solution.

The distal end of the duodenum is sutured with a continuous suture of catgut, passed right through the duodenum immediately below the forceps, and then over the forceps until the suture has been completed. The clamp is slipped off and the two ends of the suture tied together and cut short, thus closing completely the open end of the duodenum. Round the stump a purse-string suture of linen thread is passed. The end of the duodenum is now invaginated and the purse-string suture tied and cut short. Over this suture a single Halsted stitch is passed. After this stitch has been tied the needle is passed through the sheath of the pancreas, and another knot is made. Thus the stump is buried against the head of the pancreas, a manœuvre suggested by Dr. W. Meyer. The blind end of the duodenum closed in this manner rarely leaks. Still it does so occasionally, and as I prefer to take no risks, I leave a small drainage tube from the upper end of the wound down to the duodenal section. In my early cases I did not drain. In two of them leakage occurred, but fortunately the duodenal contents found their

PLATE XXXIII.

FIG. 40.—Portion of stomach usually resected, showing the dissection
of the glands. (W. J. Mayo.)
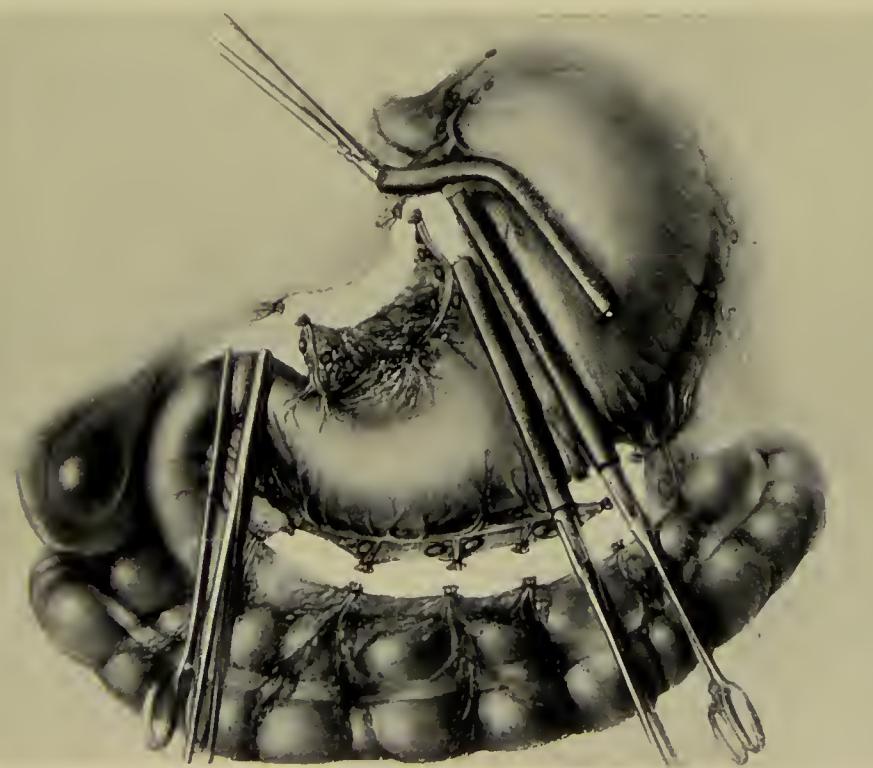
FIG. 41.—Clamps placed on stomach and duodenum isolating pyloric
end and lesser curvature. (W. J. Mayo.)

way through the upper part of the wound, and both patients recovered.

(x) The stomach is drawn downwards and to the right, and a long pair of clamps is placed on it half an inch to the cardiac side of the line of proposed section. Usually it is recommended that clamps covered with rubber tubing should be used. I prefer naked clamps, as they are not so apt to slip, and if not pressed too tightly, they have no injurious effect on the gastric tissue. A second pair of clamps is placed on the stomach distal to the first pair and at an interval of half an inch. A small pair of Porrier's forceps is placed close to the first clamp on its distal side at the lesser curvature, and a second pair at the greater curvature.

These forceps are to prevent the possibility of retraction of the stomach from the clamp when the section is made.

The stomach is now divided with a knife close to the distal pair of clamps, and both cut surfaces are cauterised with the actual cautery. The cut surface of the portion of the stomach to be removed is covered with moist gauze. Commencing at the lesser curvature, a continuous suture of linen thread is passed through all coats of the stomach. This suture should be passed at intervals of about three-sixteenths of an inch, and must be pulled tightly so as to arrest all bleeding.

Before the ends are cut short, the clamp is removed and the cut surface inspected to see if there be any vessels which require ligation.

Another clamp, covered with rubber, is placed on the stomach at a distance of one inch from the suture line. This is used to steady the stomach while the peritoneal suture is being passed. It is most important to secure complete inversion of the cut end of the stomach at the lesser curvature and at the greater curvature. This is accomplished with greater certainty if the peritoneal suture

be commenced, *not* at either of the ends, but at the middle of the remaining portion of stomach.

Therefore, I commence the peritoneal suture at the middle of the stomach. This suture is of linen thread, and is passed by the Cushing method, and inverts completely the cut mucous surfaces. If there be much tension, an additional Halsted mattress suture at each end of the suture line gives greater security. All soiled pads are now removed.

(xi) The portion of the stomach which has been separated is now turned over to the right, so that any posterior adhesions may be dealt with deliberately under direct inspection, instead of by touch alone, as would be the case if they were dealt with at an earlier stage of the operation. If the growth be adherent to the pancreas, it is better to shave off a piece of the pancreas, rather than to separate it by tearing. If the pancreas be injured, the raw surface should be covered over with peritoneum as completely as possible, and of course in such a case drainage is imperative.

The retro-pyloric glands are dissected off so that they remain attached to the stomach.

The growth and attached stomach and duodenum are now taken away, all pads removed, and the operation area sponged dry. The gastro-jejunostomy should be inspected, as possibly the resection may render it advisable to correct a kink, by the insertion of an additional suture.

(xii) The abdominal wound is closed in the usual manner, a drain to the blind end of the duodenum being brought out at the upper angle of the wound.

The steps of the operation may be recapitulated as follows:

(i) Making an opening into the gastro-hepatic omentum in order to make an exploration of the growth.

(ii) The performance of gastro-jejunostomy.

(iii) The ligation of the gastric artery.

PLATE XXXIV.

FIG. 42.—Restoration of intestinal continuity by an anterior
gastro-jejunostomy.   (W. J. Mayo.)

(iv) The ligation of the pyloric artery.

(v) Dissection of the gastro-hepatic glands from off the stomach at the pyloric and cardiac end of the lesser curvature.

(vi) The ligation of the left gastro-epiploic vessels and of the gastro-colic omentum.

(vii) The dissection of the sub-pyloric group of glands, and freeing of the inferior border of the duodenum.

(viii) The freeing of the posterior surface of the duodenum.

(ix) Division and suture of the duodenum.

(x) Division and suture of the stomach.

(xi) Separation of the posterior adhesions of the growth.

(xii) Closure of the abdominal wound.

The important points are :

1. The use of carefully placed pads to prevent contamination of the peritoneum.

2. The use of clamps with a similar object.

3. Preliminary ligation of the blood-vessels to prevent loss of blood.

4. Complete dissection and removal of all infected glands in one mass, with the portion of stomach removed.

5. Disinfection with the cautery and perfect invagination of all mucous surfaces.

(b) **Complete Gastrectomy.**—Occasionally, a case is met with in which, although the whole stomach is infiltrated with cancer, there is an absence of metastatatic deposits and of extensive glandular infection. In such cases the whole stomach may be removed, and the jejunum united to the œsophagus.

The technique is similar to that of partial gastrectomy, except that the jejunum is anastomosed to the anterior wall of the œsophagus instead of to the stomach. The anasto-

mosis is facilitated by passing a large-sized œsophageal tube from the mouth into the stomach.

8. EXCISION OF GASTRIC ULCERS

The method to be adopted varies with the position of the ulcer. In ulcers near the pylorus, the affected portion is excised, the cut ends of the duodenum and stomach closed by suture, and the continuity of the alimentary canal restored by the performance of gastro-jejunostomy. This treatment of calloused ulcers near the pylorus is known as Rodman's operation. The procedure is the same as that described under partial gastrectomy, except that the operation is much less extensive, the excision being limited to the ulcer area.

In ulcers of the anterior wall, the stomach is clamped on either side of the ulcer, two small holes being made into the gastro-colic omentum for the passage of the clamps ; an incision is made in healthy stomach wall encircling the ulcer, which is then cut out, and the gap left is sutured by means of two continuous sutures, an inner including all coats of the stomach and an outer serous suture. The abdomen is closed without drainage.

In saddle ulcers of the lesser curvature a V-shaped incision, having its base at the lesser curvature, is made through the gastric wall so as to include the ulcer and a small margin of healthy stomach. As a rule, a similar incision has to be made on the corresponding part of the posterior gastric wall, a hole being made in the gastro-hepatic omentum in order to expose the posterior aspect of the stomach. The edges of the stomach are sutured in the usual way.

Excision has been practised for hour-glass stomach. Clamps are applied on each side of the constriction. The whole of the portion of the stomach which is the seat of induration is cut away, and the two halves of the stomach united by two continuous sutures. It is advisable in these

PLATE XXXV.

FIG. 43.—Trans-gastric excision of calloused ulcer of the posterior wall of the body of the stomach. The anterior wall of the stomach is opened and the ulcer exposed. The dotted lines show site of proposed excision. (W. J. Mayo.)

FIG. 44.—Ulcer excised and through-and-through mucous suture partially completed. (W. J. Mayo.)

cases to pass a tube into the lesser sac for drainage in case of leakage.

For ulcers of the posterior gastric wall, trans-gastric resection was suggested and first performed by Dr. Lewis Pilcher. The method has been advocated by Dr. W. J. Mayo. The steps of the operation are described by Dr. W. J. Mayo as follows :

" The gastro-hepatic and gastro-colic omenta are opened above and below the ulcer. Gauze protection is introduced, adhesions are carefully separated, and if possible, the ulcer surface is cut free from the posterior attachments without opening the stomach. A piece of gauze is packed into the denuded area behind, and in all but one of our cases this temporary pack was adequate to stop hæmorrhage without the ligation of vessels. The anterior wall of the stomach is opened, and with the fingers behind, the entire ulcerated surface is pressed through the anterior incision and the ulcer excised. The gap is sutured with through-and-through sutures of chromic catgut from the mucous side transversely, and this suture line is further protected by several mattress sutures of linen, applied from the mucous side to prevent separation due to the early absorption of the catgut. The anterior wall of the stomach is then closed. Several rubber-tissue drains are carried down behind the stomach and brought out at the upper end of the abdominal wound as a safeguard."

9. JEJUNOSTOMY

Indications :

(i) Pyloric stenosis, with extreme infiltration of the stomach by cancer, and glandular infection rendering the case unsuitable for resection, or for the performance of gastrostomy or gastro-jejunostomy.

(ii) Cicatricial contraction of the stomach due to the action of strong acids or alkalies.

The operation rarely is indicated, and seldom is justi-
fiable. When the patient is in such a condition that this
operation is the only surgical treatment possible, it is scarcely
worth doing. Life can be prolonged only for a few weeks.
It is better to give the patient morphia and make the end as
comfortable as possible.

When, however, for special reasons, such prolongation of
life is highly important, the operation may be done. The
best modification of the operation is that of Mr. Mayo
Robson. The following is his description :

" The operation consists in taking a loop of the beginning
of the jejunum just sufficiently long to reach the surface
without tension ; the two arms of the loop are short-cir-
cuited about three or four inches from the surface, the short
circuiting being done either by means of sutures around a
decalcified bone bobbin, or by suture alone ; personally I
prefer the former.

" A small incision is then made into the top of the loop
just large enough to admit a No. 12 Jaques catheter, which
is inserted and passed for three inches down the distal arm
of the loop ; this is fixed to the margin of the incision in
the gut by a silk or Pagenstecher suture, and the entrance of
the tube into the bowel is further guarded by two purse-
string sutures, one over the other. The top of the loop is
fixed to the skin by one or two stitches and the wound
closed. The patient can then be fed at once with some
peptonised milk and brandy. The whole operation can be
done in from fifteen to twenty minutes, and with very little
visceral exposure.

" Should the patient be too ill to bear the little extra
time occupied by the short circuiting, the tube may be in-
serted as directed, and surrounded by two or three purse-
string sutures, a proceeding which can be accomplished in
a few minutes. In this case the loop of the bowel must not

be brought to the skin, but had better be fixed by sutures to the peritoneal margin and the aponeurosis, in order to leave part of the lumen of the attached loop within the abdomen for direct passage onwards of the intestinal fluid with the bile and pancreatic secretion."

10. GASTROPEXY

Gastropexy has been practised for the relief· of gastroptosis. The operation consists in raising the dropped stomach and fixing it higher up in the abdomen. Various methods have been adopted. Thus, Duret fixed the lesser curvature to the anterior parietal peritoneum. Rovsing fixed a considerable portion of the stomach to the anterior abdominal wall, by passing several sutures transversely through the outer coat of the stomach, then passing the sutures through the abdominal wall and tying them over a glass rod. Dr. Coffey supports the stomach by suturing the omentum along the greater curvature to the abdominal wall above the umbilicus. Dr. Beyea raises the stomach by plicating, and so shortening the gastro-hepatic and gastro-phrenic ligaments by three rows of interrupted sutures.

Probably the best method is the modification of Dr. Beyea's operation, suggested by Sir Frederic Eve. It is described thus :

" A sandbag is placed beneath the lower part of the thorax as for operations on the liver ; the abdomen is opened to the left of the middle line, and the liver well raised up and held by an assistant, the lesser omentum being thus fully exposed. If the thorax is very long and narrow a head-lamp is advisable. The stomach is suspended by four or five interrupted silk sutures passed above and through the upper part of the gastro-hepatic omentum, and below through the lesser curvature.

" Advantage is taken of the fact that ·the gastro-hepatic

omentum close to its attachment to the diaphragm and transverse fissure of the liver is much thicker than lower down near its attachment to the stomach ; but if the whole membrane is equally thin the sutures are passed above through the liver substance itself, just anterior to the transverse fissure. This is rarely necessary. Below, the sutures are carried through the serous and muscular coats of the stomach just in front of the attachment of the lesser omentum, the vessels, of course, being avoided. The suture furthest on the right is not placed quite up to the pylorus, in order that when the sutures are tied the pyloric end of the lesser curvature may be on a slightly lower level than the portion on the cardiac side.

" A very long needle-holder is required in passing the sutures above under the diaphragm."

It is very doubtful whether any operation for gastroptosis is of real value. Personally, I am strongly against operating in such cases. The patients are seldom better for it, and are often worse.

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CHAPTER IV

THE TREATMENT OF PATIENTS AFTER GASTRIC OPERATIONS

When the operation is completed, the patient should be carried back to bed with the utmost care and gentleness. Sudden lifting or jolting not only puts an unnecessary strain on the wound, but may cause the patient to vomit. The bed should be warmed thoroughly by means of hot-water bottles, but these should be removed, except one for the feet, before the patient is put back to bed. It is desirable to keep the patient warm, but not to cause perspiration. The hot-water bottles should be of stone or indiarubber, and should be enclosed in a flannel bag. The tin hot-water bottles used so commonly in hospitals are very undesirable, and sometimes cause severe and troublesome burns.

Position.—The patient should be propped up in the sitting position (Fowler's position) by means of half a dozen pillows, or a bed-rest. The best arrangement that I know of for preventing the patient from slipping down in the bed, is that devised by Sister Agnes of King Edward VIIth's Hospital for Officers. It consists of a board made of hard wood, $\frac{3}{4}$ inch thick, $6\frac{1}{2}$ inches wide, and of a length corresponding to the width of the bed on which it is to be used. At each end of this board is fixed an iron bracket, from which projects a short pin which fits into a hole drilled in the side rails of the bed. The brackets are made so that when the pins are fitted into the side rail of the bed, the board lies across the mattress inclined at an angle of 45° (Fig. 45). The patient's thighs rest against the board, on which a pillow

is placed for greater comfort. This simple contrivance can be made at a comparatively trifling cost, and is most effectual in maintaining the patient in the desired position.

In an emergency, a simple but less efficient " bed stop " may be improvised in the following manner : A stout bolster (about nine inches in diameter) is placed under the patient's

FIG. 45.—Sister Agnes's " Bed Stop."

thighs. To each end of the bolster a piece of stout webbing is fixed. By tying the other ends of the webbing to the head of the bed, the bolster is fixed, so that it prevents the patient from slipping. The bolster is more efficacious if it be stiffened by passing a broomstick through its centre from end to end. The webbing in this case is fixed to the ends of the broomstick instead of to the bolster. The disadvantage of this method is that the webbing stretches, and so allows the patient to slip down. To some extent

this drawback may be obviated by using leather straps instead of webbing to fix the bolster. If webbing straps be used, the arrangement at best is a makeshift.

At night the patient may be turned partially on one side, and after three or four days may, if preferred, lie down on one or other side during the night. I think the Fowler position is the best, after all abdominal operations, but is indicated especially in cases of septic peritonitis and suppurative appendicitis, because it allows free fluid in the abdominal cavity to gravitate to the pelvis, and in suppurative cases pus is thus less likely to track up towards the diaphragm. This is an important matter, for it has been shown that the upper half of the abdomen is much more susceptible to micro-organisms than the lower half. To a considerable extent, the pelvic peritoneum can take up and destroy micro-organisms, but if they reach the area round the liver they become absorbed uninjured, and may cause pleurisy, empyema, and even pyæmia. If there be severe shock, it may be advisable to keep the patient lying flat for a few hours, but in all septic and suppurative cases, I have the patient propped up at once, whether shock be present or not.

Continuous Proctoclysis.—For the first forty-eight hours all my abdominal cases have continuous saline injections by the rectum. This procedure is indicated especially in cases of suppurative peritonitis, but I am satisfied that it is of high value as a routine treatment after all abdominal operations. It is commenced as soon as the patient is back in bed. The largest size rubber rectal tube should be used, with a hole at the end and two holes at the side. The tube should be passed about three inches into the rectum, and should be kept in place by a piece of strapping passed round the thigh, or one of Murphy's self-retaining tips [1] may be used.

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[1] Messrs. Allen & Hanburys have made some of these for me, and they answer very well.

The rectal tube is connected by means of two feet of large rubber tubing with a douche can containing from four to five pints of saline solution, which is kept at a temperature of 125° F. The douche can is raised from four to eight inches above the level of the bed, so that the fluid runs slowly into the rectum at the rate of a pint an hour. After 1½ pints have been given the flow should be stopped, but the rectal tube should not be removed, and the flow should be started again two hours from the time it was originally commenced. After some hours it may be necessary to diminish the rate of flow, or lengthen the interval between the administrations. In this way from ten to fourteen pints can be given in the course of twenty-four hours. The important points are : (1) to keep the temperature of the saline solution constant, and (2) to regulate the flow by gravity, and not by constricting the delivery tube. A feeling of discomfort on the part of the patient indicates that the flow is too rapid, and if the saline be not retained, generally it means, either that the flow is too rapid, or that the temperature is too high, or too low. Dr. Murphy, to whom we are indebted for this valuable method, maintains that if the saline be not retained, it is because it is not given properly. While I am not prepared to deny that a few patients do not retain salines well, I have no doubt that the successful employment of continuous proctoclysis depends mainly on the skill and attention of the nurse. Some nurses are far more successful than others. After an abdominal operation every patient should have the undivided attention of a special nurse for the first thirty-six or forty-eight hours. In a properly

FIG. 46.—The author's electrically heated saline can.

managed hospital or nursing home, this is arranged as a matter of routine. The regulation of the temperature of the saline requires constant attention, and to get over this difficulty, I have had an apparatus made which simplifies greatly the use of this method.

The apparatus (Fig. 46) consists of an electro-plated douche can which holds about five pints. In the front of the can are a thermometer and a gauge glass, by the side of which the can is graduated in half-pints, so that the amount of saline entering the rectum can be estimated readily. The saline leaves the can through a delivery-tube with a $\frac{1}{2}$-inch bore, to which is attached two feet of rubber tubing connected with a large rectal tube. Under the bottom of the can is an electric heater, which can be connected with any electric supply of suitable voltage by means of a flexible cord and wall plug. The can is suspended on an adjustable stand mounted on castors, so that it can be readily wheeled up to the bedside (Fig. 47). I have found by experiment that with a ward temperature of from 65° to 70° F. the solution in the can must be kept at a uniform temperature of 125° F. in order to ensure that the saline solution enters the rectum at a temperature of from 99° to 100°. The electric heater is so made, that if the saline solution be put into the can at a temperature of 125° F. the temperature remains almost constant so long as the current is switched on.

Continuous proctoclysis acts in three ways : (1) It dilutes
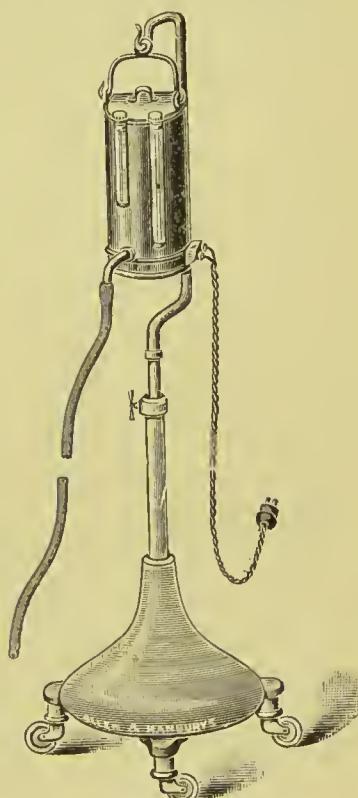
Fig. 47.—The author's apparatus for continuous proctoclysis.

toxins and renders them less harmful. (2) It stimulates the lymphatic flow and so drains the peritoneal cavity. (3) It raises the blood pressure and so diminishes shock. Incidentally thirst is relieved. In cases of septic trouble it should be continued until the patient is out of danger.

Diet.—There is still a lingering superstition that patients must be half-starved after an abdominal operation. A few months ago a surgeon writing on this subject advised nothing but water for twelve hours, and in stomach operations nothing but a little water for three days. I regard such starvation as totally unnecessary, and in old or feeble patients positively harmful. Even after operations on the stomach I begin feeding my patients at once. In one of my gastro-jejunostomies for pyloric obstruction, I allowed the patient to have two mutton chops and a milk pudding on the third day. He was thoroughly exhausted by months of vomiting, and was ravenously hungry. He thoroughly enjoyed his meal, and was all the better for it. Of course, I do not suggest this as a routine treatment, but I mention the case to emphasize how groundless is the fear of early feeding. If the anastomosis be efficiently performed, the risk of the sutures giving way may be neglected, so far as feeding is concerned.

As soon as the patient desires a drink, small quantities of hot water are given, and if this be retained one-ounce doses of milk diluted with two parts of water. The quantity is increased gradually up to two ounces hourly. I allow a cup of tea the same day as the operation if the patient wish it. After severe operations, and in elderly and feeble patients, I always order an ounce of Sanatogen to be given during the course of the twenty-four hours. I regard this preparation as a valuable restorative in these cases. On the day after the operation, Benger's food and calves' feet jelly are given as well as milk. As soon as the bowels have been opened

the patient is allowed fluid *ad libitum*, eggs, thin bread and butter (no crusts), and other soft solids, and usually ordinary diet is resumed in a week or ten days.

As a general rule the patient's inclination is a reliable guide to the quantity of food required, although in a few cases some coaxing and diplomacy are necessary to induce the patient to take adequate nourishment. It is impossible to lay down hard-and-fast rules as to feeding, and general rules have to be modified in individual cases. After operation for septic peritonitis, no food is given by the mouth until the bowels have been opened thoroughly. After gastro-jejunostomy for gastric or duodenal ulcer, especially if associated with hyper-acidity, the diet must be more limited in quality, although the quantity need not be curtailed. I always urge these patients to keep on a milky diet for at least six months. On the other hand, after gastro-jejunostomy, or partial gastrectomy for cancer, I feed the patients up more rapidly, allowing mutton or beef essence, jelly, eggs, and Benger's food on the second day, and often fish cream or chicken cream on the third day. Patients who have been exhausted by weeks or months of vomiting will not stand starvation, and their tissues possess feeble power of repair, unless they be provided with plenty of nourishing food.

Rectal Feeding.—Rectal feeding is a scientific term for starvation. Formulæ for so-called nutrient enemata composed of eggs, milk, beef-tea, &c., still survive in text-books and hospital pharmacopœias, relics of the superstitions of the past. I often wonder whether the surgeons who prescribe them have seen or smelt the odoriferous mess which is washed out of the rectum when such concoctions are administered. In my experience rectal feeding is quite unnecessary after abdominal operations, and I have satisfied myself by experiment that little, if indeed any, of the constituents of these mixtures is absorbed. After an ordinary

abdominal section, usually the patient can take enough
nourishment by the mouth, and the fluid required by the
body can be administered by the rectum in the manner
already described. In cases of septic peritonitis, as a rule,
the patients have been in good health up to the time of the
onset of the illness, and can do without much nourishment
until food can be administered by the mouth. In these
patients it is fluid that is required rather than nourishment.
In those exceptional cases in which nourishment is necessary,
and cannot be given by the mouth, I add dextrose to the
saline injection.

The Bowels.—I regard the early opening of the bowels
as one of the most important points in the treatment of
patients after abdominal operations. No hard-and-fast rules
can be laid down as to how soon an aperient should be
given. Each case must be dealt with according to the con-
ditions present, and there is no detail of after-treatment in
which sound judgment and experience are of such import-
ance. My guiding principle is, that the sooner the bowels
are opened, the sooner is the patient out of danger. In an
ordinary uncomplicated case I give an aperient usually on
the third morning, and in my opinion there is no better
aperient than calomel given in half-grain doses every hour
until the patient has had three or four grains.

A few hours after the administration of the last dose a
turpentine enema is given. If this be ineffectual, three or
four drachms of castor-oil are given at night, followed by
another enema. In pelvic cases, in cases in which there has
been much handling of intestine, or in cases in which there
is distension after operation, I commence the administration
of calomel on the second day. After operations for septic
peritonitis and for perforated gastric or duodenal ulcer
(provided I have performed a gastro-jejunostomy as well as
sutured the ulcer), I often commence the administration of

calomel the same night as the operation, in some cases as soon as the patient has come round from the anæsthetic. In these cases, in addition to calomel, sometimes I give subcutaneous injections of eserine salicylate, gr. $\frac{1}{100}$ every four hours until five or six doses have been given. When there is distension, as well as vomiting, a good plan is to give five grains of calomel immediately after an attack of vomiting. This may be repeated if necessary. In the case of a woman with extreme distension and vomiting after operation for extra-uterine gestation, nearly twenty grains of calomel were given in this way during twenty-four hours. On the third day the patient's bowels were freely opened, she experienced immediate relief, and thereafter made an uneventful recovery. I have never yet had to regret giving an aperient early.

Treatment of the Wound.—I believe in a tight bandage for the first twelve hours ; after this it may be loosened and readjusted. There is nothing so comfortable as a many-tailed bandage made of strips of domette seven inches wide and about seven feet long. The strips should be placed one inch apart, and should not be sewn together behind, as is usual. It adds to the patient's comfort if the bandage be undone and rebandaged every day, without disturbing the dressings. Every two or three days the bandage may be taken off and a fresh one put on. The old one can be washed and ironed ready for the next change. I have my wounds dressed and the skin suture removed on the sixth day. The sutures used to anchor the dressing are removed a few days later.

Drainage Tubes.—Drainage tubes occasionally are left in too long, but far more frequently they are removed too soon. This is especially the case after operations for general suppurative peritonitis, in which it has been necessary to place a drainage tube in Douglas's pouch. Such a tube should be kept *in situ* for at least a week, and sometimes for longer. If removed earlier it may be impossible to replace it, and as

a result of too early removal, an abscess may form which may require another operation for the evacuation of the pus. As a general rule a drainage tube should not be removed until any discharge of pus has ceased for three or four days.

The Duration of Rest in Bed.—There is a great diversity of opinion as to how long a patient should be kept in bed after an abdominal section. Personally, I believe in shortening rather than in lengthening this period. My practice varies according to the situation of the wound. After operations for appendicectomy, in which I use the Battle incision, I allow the patient to get on to a couch in a week, if so inclined, and to sit in an armchair two or three days later. When the incision is above the umbilicus, three weeks in bed, and when below the umbilicus a few days longer, are advisable. In operations for cancer I insert some additional through-and-through sutures, and then get the patient into a chair on the fourth or fifth day. Patients suffering from cancer do not bear confinement to bed well, and the sooner they are got up the better. After operation for gastric ulcer in patients who have been losing blood, or who have been on a restricted diet for a long time, the decision as to when it is advisable to allow them to get up needs some discrimination. Often such patients have weak, flabby hearts, and require feeding up, and a course of digitalis and iron, before they are fit for much movement. If they be allowed up too soon, they may die quite suddenly from syncope. In such cases, a subnormal temperature and a slow pulse should be regarded as danger signals. On the other hand, I believe that too long confinement to bed favours thrombosis, with the danger that when the patient gets up an embolus may be detached.

All patients should wear a firm binder or abdominal belt for six months after operation, and before discarding the belt should have abdominal massage for two or three weeks.

CHAPTER V

COMPLICATIONS AFTER GASTRIC OPERATIONS

1. **Shock.**—For practical purposes we need not draw a distinction between shock and collapse. The treatment is the same, namely, the treatment of " the condition resulting from a fall in the general blood pressure." The chief causes of shock after an abdominal operation are : 1. A prolonged operation. 2. Hæmorrhage. 3. Exposure or rough handling of viscera.

There is another type of shock due to the absorption of toxins, which is apt to occur after operations for suppurative peritonitis. This is the most serious type of shock, and the most difficult to treat, and I am convinced, contrary to the opinion expressed by some, that washing out the peritoneal cavity greatly increases its severity.

The ideal treatment of shock is preventive. In my judgment by far the most potent means of preventing, or at any rate lessening, shock is the use of morphia and ether instead of chloroform for the production and maintenance of anæsthesia. When ether is given by the open method it is rare for symptoms of shock to be marked, owing to the action of this combination of narcotics in blocking the afferent nerve impulses. I do not say that shock is absent entirely, but that in the cases in which it occurs, its onset is delayed until the effects of the ether and morphia are passing off, and at this stage it is easier to treat shock than during, or just after, the operation.

The first thing to bear in mind in the treatment of shock

is that strychnine, brandy, and other so-called stimulants
are useless, and indeed positively harmful. This has been
shown conclusively by the work of Dr. G. W. Crile and
others. Strychnine given to a healthy person raises the
blood pressure, but when administered to a person already
suffering from shock, it causes a marked fall of blood pressure,
a result exactly opposite to that required in the treatment
of shock. The best treatment for shock is the administra-
tion of continuous saline solution by the rectum, by the
method described already. As regards drugs, adrenalin
causes a marked rise in the blood pressure. A drachm of
adrenalin chloride (1 in 1000) may be added to each pint of
saline solution. The objection to adrenalin is that its effect
is very transient, so that to be of service the administration
of the drug must be continued. Extract of pituitary body is
a much more powerful agent, and its effects are more lasting.
In an adult, an injection into the deltoid muscle of seventeen
minims (1 c.c.) causes a marked rise in the blood pressure,
which lasts for eight or ten hours. It is, however, a powerful
drug, and the dose must be regulated carefully according to
the age and strength of the patient. Until shock has passed
off, the patient's head should be kept low by raising the foot
of the bed. The repeated application to the head and fore-
head of cloths wrung out in very hot water is a valuable
addition to the methods for treating shock, which I have
mentioned.

I never give intravenous injections of saline solution,
as I believe that in some cases they do harm. If there be
any difficulty with rectal administration, I prefer to let the
saline solution run into the subcutaneous tissues of the
breast and axilla. As I have mentioned already, I have all
suppurative cases propped up in bed at once, whether shock
be present or not.

2. **Pain.**—I have included pain among the complications

of abdominal operation, because I believe that pain as distinct from discomfort is the exception rather than the rule. There is no doubt that patients vary very much in their susceptibility to pain, and in their capacity for bearing it. It is rare for hospital patients to complain much of pain after an abdominal operation; private patients, on the other hand, complain much more frequently of pain during the first twenty-four hours. I am very strongly averse from giving morphia after an abdominal operation; it relieves pain, but it is harmful in other ways, and certainly adds to the patient's subsequent discomfort. We should try to ascertain the cause of the pain, and if possible treat such cause. Pain may be due to the patient's position, to undue tightness of the bandage, to excessive tightness of sutures, and in wounds above the umbilicus to the movements of the diaphragm. A frequent cause of pain, especially after the first twenty-four hours, is intestinal colic. This can be relieved at once by a turpentine enema, or a dose of calomel. In such a case morphia is the very worst mode of treatment. I confess that I dislike giving any drugs after an abdominal operation, but if pain be so severe as to require treatment, ten grains of aspirin repeated every six hours up to thirty grains, or a dose of trional, is the least harmful remedy. In neurasthenic patients, the value of an hypodermic injection of sterile water should not be overlooked. Often it acts like a charm.

3. **Septic Peritonitis.**—It is scarcely an exaggeration to say that septic peritonitis as a sequence of operation in a clean case has been abolished by the methods of modern aseptic surgery. It may occur from leakage owing to faulty application of the sutures of an anastomosis. With a double row of sutures carefully applied, leakage should not occur, and I have not yet met with it. Leakage may occur in rare instances through no fault in technique, but owing to

feeble repair on the part of the patient. Septic peritonitis may result also from contamination of the peritoneum with intestinal contents at the operation. By the use of clamps and pads such soiling is reduced to a minimum. Thorough preparation of the patient beforehand, on the lines laid down in a previous chapter, greatly reduces the probability that such soiling as occurs will do serious harm. The infection produced by the soiling will be slight, and the peritoneum is able to deal with it safely.

The treatment of septic peritonitis following operations is beset with difficulty. In by far the larger majority of the cases, the safest course is to treat the patient by absolute rest, lavage of the stomach, and ice to the abdomen, in the hope that the peritonitis may be limited. If it be probable that leakage has occurred, the patient's only chance is a second operation, to repair the leak, to cleanse the surrounding parts, and to establish drainage. Such operations rarely are successful. The patient's strength and nerve-cells have been exhausted already by one operation, and will be depleted still further by another. Therefore, before deciding to reopen the abdomen, the surgeon should have strong grounds for believing that a leakage has occurred. It is only under such circumstances that a second operation is justifiable.

When septic peritonitis is present at the time of the original operation, early opening of the bowels is indicated. In my experience, when once the bowels are thoroughly well opened, the patient may be regarded as out of danger.

Our aim should be to prevent peritonitis; strict asepsis, the use of clamps and pads to prevent the soiling of the peritoneum, and early opening of the bowels after operation, are the chief safeguards against this complication.

4. **Post-anæsthetic Vomiting.**—When ether by the open method, in conjunction with morphia, is the anæsthetic em-

ployed, post-anæsthetic vomiting is rare as a complication of abdominal operations. Since this anæsthetic has been the one usually adopted for my patients, I do not think that I have had to treat a single case of post-anæsthetic vomiting. If vomiting, due to the anæsthetic, persist, the best plan is to give a good drink of warm water containing 20 grains of bicarbonate of soda. If this fail, the stomach may be washed out. Vomiting is certainly less common when the patient is propped up in the sitting position. Care should be taken that the head be turned to one side by placing a pillow under the opposite shoulder. The persistent vomiting which sometimes follows chloroform narcosis, may be relieved, in some instances, by adding an ounce of glucose to the saline injection. In operations for intestinal obstruction, often it is advisable to wash the stomach out before the anæsthetic is given, and in all such cases it is well to do so before the patient is put back to bed after the operation. In cases of vomiting from septic peritonitis, nothing should be given by the mouth, gastric lavage should be repeated at regular intervals, and the administration of calomel and eserine salicylate should be commenced at once in order to secure emptying of the intestinal canal as early as possible. When this has been accomplished usually the vomiting ceases.

5. **Pseudo-ileus.**—Pseudo-ileus, or paralytic distension of the intestine, is most likely to occur in cases in which there is suppurative peritonitis, or in which there has been much handling, damage, or exposure of the intestines, or in cases in which it has been necessary to pack part of the abdominal cavity with gauze. If calomel be administered early, as I have advised, this complication will not occur often. When it does occur, the treatment is energetic administration of eserine salicylate, calomel, and turpentine enemata. I confess, however, that I am very sceptical as to the much-vaunted efficacy of eserine salicylate. When gauze packing

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has been employed, the packing should be removed as early as possible. Strychnine is strongly recommended by some for the treatment of pseudo-ileus. In my experience it has proved quite valueless.

6. **Lung Complications.**—Bronchitis and pneumonia follow abdominal operations with far less frequency than formerly was the case. Often they are attributed to the anæsthetic, especially to the chilling and irritating effect of ether vapour on the respiratory passages.

I confess I am very sceptical as to the occurrence of a true ether pneumonia. Mr. Bellamy Gardner has shown that when ether is being given by the open method, the temperature of the vapour under the mask is only one degree below that of the surrounding atmosphere. Dr. W. J. Mayo has told me that in more than one instance he has seen pneumonia follow a gastric operation performed under local anæsthesia.

Dr. W. Pasteur has shown that the incidence of lung complications is greater after chloroform than after ether, the percentage in the case of chloroform being 7·74, and in the case of ether 5·18. Possibly in many instances lung complications have been due directly to the rebreathing of the contaminated air in the bag of a Clover's inhaler, an apparatus which has served a useful purpose in the past, but which, now that we have the open method of ether administration, should be seen only in museums of medical antiquities. I think there can be no doubt that the adoption of the Fowler position after abdominal operations has diminished greatly the frequency of lung complications. Formerly pleurisy and empyema were not infrequent after operations for acute appendicitis. Since the routine use of the Fowler position I do not remember that I have met with either of these complications. Probably in some instances lung complications are due to slight local sepsis resulting in

the detachment of a small septic embolus. In by far the greater number of cases I believe bronchitis and pneumonia following operation are due to deficient aëration of the bases of the lungs and to the accumulation of secretion in these situations. This hypostatic congestion is the result of the interference with the working of the diaphragm, partly from too tight bandaging, and partly from the involuntary inhibition of its movements caused by the wound.

It is no doubt a truth, based on the observations of many surgeons, that lung complications follow gastric operations more frequently than any others. And it is in stomach operations that the wound in the abdominal wall is situated nearest to the diaphragm.

I feel convinced that the occurrence of pneumonia and bronchitis in my practice has been diminished greatly by encouraging the patient to take deep breaths at regular intervals during the days following operation.

In the prevention of lung complications I attach great importance to keeping the patient warm during the operation, both by means of clean, warm clothing, and by the use of an operation table heated by electricity or other means.

The treatment of the bronchitis and pneumonia after abdominal operations presents no unusual features.

7. **Parotitis.**—Parotitis may occur in cases in which food by the mouth is withheld. The treatment is preventive, with careful attention to the cleansing of the mouth, and frequent rinsings with listerine or hydrogen peroxide. In addition, the plan adopted by Dr. Soltau Fenwick is a good one. He gives the patient a baby's rubber teat to suck. The constant sucking induces a flow of saliva which helps to keep the mouth clean. The teat should be sterilised frequently by boiling. When parotitis occurs, fomentations should be applied, and if pus form, it should be evacuated in the ordinary way.

8. **Phlebitis and Thrombosis.**—Phlebitis and thrombosis are less common after gastric than after pelvic operations. Thrombosis usually is secondary to phlebitis, but occasionally thrombosis is the primary condition. It is more likely to occur in feeble, debilitated patients whose blood current is sluggish. One of my patients died quite suddenly three days after an ovariotomy. At the post-mortem examination an ante-mortem clot was found in the pulmonary artery. Phlebitis is probably septic in origin, and is met with most frequently in the saphena and femoral veins of the left lower limb. It is rare for this complication to occur before the second or third week.

The treatment is absolute rest, and elevation of the affected limb, and the period of confinement to bed should be increased to five or six weeks. Sir Berkeley Moynihan speaks well of the intravenous injections of collargol.

9. **Post-operative Hæmatemesis.** — Hæmatemesis may occur after operations on the stomach owing to imperfect application of the sutures, so that bleeding ensues from the cut edges of the stomach, or intestine. This may be spoken of as avoidable post-operative hæmorrhage, in distinction from unavoidable post-operative hæmatemesis.

By unavoidable post-operative hæmatemesis is meant, hæmorrhage for which there is no apparent cause. Although many papers have been written on the subject, the ætiology of such hæmorrhage is not clear. Professor Von Eiselsberg considers that it is due to injury of the omentum. This certainly does not explain all cases. Dr. Rodman attributes it to sepsis, an explanation doubtless applicable in some cases, but not in others. It is a very fatal complication, with a mortality rate, according to Dr. Powers, of nearly 70 per cent. I have met with one instance, after separating adhesions around a jejunal ulcer. The patient had considerable hæmatemesis on the second and third days, but recovered.

The amount of blood vomited at one time is small, but the vomiting is frequently repeated. Surgical intervention is contra-indicated in these cases. The treatment should be ice to the abdomen, gastric lavage, very hot saline by the rectum, and abstinence from food by the mouth and the administration of calomel.

10. **Acute Dilatation of the Stomach.**—Acute post-operative dilatation of the stomach is a serious complication. In some cases the gastric dilatation is the result of septic peritonitis, and is accompanied by a general distension of the intestines. In other cases the stomach alone is dilated. Why dilatation occurs is not quite clear. Allbrecht suggested that the superior mesenteric artery presses upon and causes obstruction of the third part of the duodenum. This explanation doubtless holds good for some cases. Other writers maintain that the dilatation is the result of a primary paralysis of the musculature of the stomach. Dr. Box and Mr. Wallace suggest that two factors come into play in producing acute gastric dilatation. " First, a paralytic condition of the viscus which leads to distension and then, at a certain stage, the distended stomach actually produces obstruction by pressing on the duodenum on the front and to the left of the spinal column." An excellent account of the subject has been published by Dr. Campbell Thomson.

Repeated vomiting is the prominent symptom, the vomit being brought up in gulps without straining, and at frequent intervals. When, after an abdominal operation, vomiting persists or comes on without apparent reason, the dressings should be undone and the abdomen examined. The bulging of the abdomen above the umbilicus, and retraction below, are very striking in cases with this complication. As to treatment, the stomach should be washed out at intervals, and the patient placed in the knee and elbow position. In the majority of instances, unless the dilatation be due to

septic peritonitis, these means will suffice. By keeping the stomach empty by lavage, gradually it recovers its tone and is able to empty itself. Acute dilatation may occur after any abdominal operation, especially after operations for perforated gastric or duodenal ulcer ; therefore a close watch should be kept for this complication, as sometimes a fatal issue may be prevented by timely lavage.

I have met with this complication four times. In one of them I performed gastro-jejunostomy with a fatal result. I think gastro-jejunostomy is contra-indicated in these cases, as with the existence of gastric atony, the short circuit is of little use. In three of my cases, acute dilatation followed gastro-jejunostomy ; two of them were after operation for perforated duodenal ulcer, and in the third after operation for " trifid stomach." In the latter case the patient vomited profusely for sixteen hours and I was not informed of her condition. When I saw her she was cold and clammy, the pulse almost imperceptible. I drew off nearly five pints of dirty fluid from the stomach. I did not expect her to recover. To my surprise, after being very ill for twenty-four hours, she made an uneventful convalescence.

Complications peculiar to Gastro-jejunostomy

(a) **Immediate.**—The condition known as " circulus vitiosus," or regurgitant vomiting, was formerly the most dreaded complication of gastro-jejunostomy. The vomiting was attributed to the reflex of bile into the stomach from the afferent loop of the jejunum. The presence of bile in the stomach has no injurious effect, provided it can get out again. It is not the presence of the bile, but the obstruction to its leaving the stomach, which causes the vomiting. Various explanations have been given as to the cause of regurgitant vomiting. Some believe that it is due mainly

to the leaving of a loop of jejunum between the flexure and the anastomosis, and that this loop, becoming "water-logged," is unable to pass on its contents, or by its weight causes a kink at the site of the anastomosis. Why a piece of healthy intestine should become water-logged, apart from mechanical obstruction, is not explained. There is not, I think, any evidence of such a causation. I believe that most, if not all, cases of regurgitant vomiting are due to mechanical obstruction at the efferent opening.

As Dr. Terrier has remarked : " The establishment of the circulus vitiosus is due to faulty operating." Whether the vomited material regurgitate from the stomach into the afferent loop, and thence back to that organ through the pylorus, is immaterial ; the real factor is obstruction at the efferent opening, and when water-logging of the jejunal loop occurs it is secondary to some obstruction.

After the anterior operation, obstruction may be caused by a kink at the point where the efferent limb is attached to the stomach, or by the use of a jejunal loop of insufficient length to allow of its being brought over the transverse colon without tension.

After the posterior operation, I am of opinion that in many of the cases, obstruction is due to accidental twisting of the jejunum on its longitudinal axis during the process of suturing, or to flattening of the jejunum by the inclusion of too much tissue in the sutures.

Dr. W. J. Mayo has pointed out that the normal direction of the jejunum at its origin is from right to left. If, then, the jejunum be attached to the stomach iso-peristaltically, a sharp angle is produced immediately above the anastomosis, which may give rise to trouble.

In the majority of cases gastric lavage for a few days suffices to cure the patient.

Dr. P. Mauclaire states that the knee-elbow position for

twenty-four hours is sufficient to arrest the symptoms in many instances. If, in spite of these measures, serious vomiting continue, the abdomen must be reopened. If an anterior gastro-jejunostomy has been performed the surgical treatment is very simple; an entero-anastomosis is performed between the afferent and efferent limbs. I have never had occasion to treat a case of acute regurgitant vomiting after the posterior operation, but the performance of an entero-anastomosis in these cases must be a very difficult proceeding.

I believe that some of the slighter cases of regurgitant vomiting, which do not follow operation immediately, are due to simple constipation. One of my own patients returned two months after gastro-jejunostomy, and stated that about once a fortnight she had severe attacks of vomiting. By inquiry I elicited the fact that the attacks invariably were preceded by constipation for several days. The patient was instructed to pay great attention to obtaining daily evacuation of the bowels, and since that time she has had no further trouble. I have observed two other similar instances. Why after gastro-jejunostomy should constipation give rise to vomiting? I think that a simple explanation may be offered. It is probable that when any part of the intestine is loaded with fæces, the onward peristaltic waves are arrested by the distension and temporary paresis of that portion of the gut. In such a case a reflux wave of peristalsis may occur, tending to drive the intestinal contents backwards. Now, in a normal individual, probably the duodenum acts as a syphon-trap, and so checks regurgitation of intestinal contents into the stomach from such slight reflux movements as would be caused by temporary obstruction. This syphon action of the duodenum is further aided by the contraction of the pylorus, so that little or none of the intestinal contents gains entrance to the stomach. After gastro-jejunostomy, however, both the syphon-trap and the

pyloric sphincter are put out of action, so that there is no obstacle to regurgitation from the efferent loop into the stomach. In this way a degree of constipation, which in a normal individual would cause discomfort only, may produce vomiting in a person upon whom gastro-jejunostomy has been performed. I think that this is the explanation of the so-called " bilious attacks " from which patients suffer sometimes after gastro-jejunostomy.

After gastro-jejunostomy there is always a slight regurgitation of bile into the stomach. In some instances this is excessive. The patient does not vomit, but is uncomfortable, has a feeling of nausea, and suffers from loss of appetite. In some of the cases the fault is one of technique, a slight obstruction at the anastomosis, but not sufficient to cause vomiting. With modern technique this ought not to happen. More often the reason is that gastro-jejunostomy has been performed for gastric symptoms in patients who had no gastric lesion. If the patient have some other lesion, such as gall-stones, or appendicitis, gastro-jejunostomy will not give relief. If gastro-jejunostomy be performed on a markedly atonic or gastroptosed stomach, the result almost certainly will be a failure. I have opened the abdomen in many instances in which symptoms have persisted after gastro-jejunostomy has been performed elsewhere, and have found no lesion present which rendered the anastomosis necessary. In these cases undoing the gastro-jejunostomy and restoring the continuity of the intestinal canal usually has given complete relief. In some of them either a diseased appendix or cholelithiasis was discovered and dealt with.

Diarrhœa.—Cases have been recorded in which death has resulted, apparently from simple diarrhœa. Attention has been called to this complication by Kelling, Anchütz, and by Carle and Fantino, who state that of eleven fatal cases

of gastro-jejunostomy, in four the cause of death was diarrhœa. I have observed this complication twice, but in neither instance did it cause any alarm. I believe that it is probably a symptom of intestinal toxæmia, and is an indication for the administration of calomel. In my first case, after gastro-jejunostomy for ulcer, it was checked at once by the administration of two doses of calomel and a large turpentine enema. In the other case, after gastro-jejunostomy for advanced carcinoma, it proved more troublesome, but ceased gradually after the patient was given one-sixth of a grain of calomel four times a day.

Internal hernia.—Several instances of internal hernia after gastro-jejunostomy have been recorded. In most of them a portion of small intestine has slipped through the artificial aperture in the mesocolon and has become strangulated.

So far as I am aware, since the adoption of the practice of suturing the edges of the mesocolon to the jejunum or stomach, no instance of this complication has been recorded.

(b) **Remote.**—The complication of jejunal and gastro-jejunal ulcer is dealt with in a later chapter (Chap. VII).

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CHAPTER VI

THE PHYSIOLOGICAL EFFECTS OF THE OPERATION OF GASTRO-JEJUNOSTOMY

THE correct application of any surgical procedure depends on a proper appreciation of its effects. It is a truism that it is unscientific to perform any operation when the indications are not clear, and the precise effect to be aimed at is uncertain. Therefore, from a clinical standpoint, it is important to answer the question, how does gastro-jejunostomy act ? Is the operation a mechanical one, or has it any physiological effect on the gastric functions ?

The prevailing view appears to be that the operation is a purely mechanical one, the provision of a new opening into the intestine allowing the food to pass out more readily and more quickly, and, in the case of a duodenal ulcer, diverting the food from passing over the ulcer. For a good many years I have been advocating the view that the operation of gastro-jejunostomy is a physiological one. Sir Berkeley Moynihan, on the other hand, has expressed his belief that the " physiological explanation of gastro-jejunostomy is rot."

It is difficult by any mechanical explanation to account for the beneficial effects of gastro-jejunostomy in the absence of pyloric stenosis. Take the case of gastro-jejunostomy for duodenal ulcer. The view that a gastro-jejunostomy acts by preventing the food from passing over the ulcer is no longer tenable. We know from the evidence of radiography that in some cases the food continues to leave the

stomach by the pylorus. If the mechanical explanation of the action of gastro-jejunostomy be correct, such cases would not be benefited by the operation. This I believe is contrary to experience. My view is, that it is immaterial whether the food leaves the stomach by the pylorus or by the stoma, although in the majority of cases the food leaves the stomach mainly by the anastomotic opening. If the mechanical explanation of gastro-jejunostomy be correct, the pylorus should be occluded deliberately in every case when performing gastro-jejunostomy. Some surgeons do this, but so far as I am aware there is no evidence that warrants the conclusion, that the results in cases in which the pylorus has been occluded, are better than in those in which this has not been done.

Take the case of a gastric ulcer in the body of the stomach at the lesser curvature or near the cardia. How does the mechanical explanation of gastro-jejunostomy explain the relief of pain in these cases ? The anastomosis cannot prevent the contact of food with the ulcer ; nor does it, as I shall show presently, hasten invariably the evacuation of the stomach.   There are some who deny that a gastro-jejunostomy is of any value in cases in which the ulcer is not near the pylorus.   I hold a strong opinion to the contrary. I have performed a large number of gastro-jejunostomies for ulcer of the body of the stomach, and have followed up the after results with great care, and in two cases only has the result been unsatisfactory, and in one of them the patient died several years later from cancer of the stomach, which arose quite independently of the original ulcer.

The Presence of Bile in the Stomach.—The presence of bile as demonstrated by Gmelin's reaction was detected in the gastric contents after a test meal in 73 per cent. of my patients after gastro-jejunostomy. It is obvious that if bile gain an entrance into the stomach pancreatic secretion

must do so as well. That Gmelin's reaction was present in 73 per cent. only of the cases, does not prove that bile was not present in the gastric contents of the other 27 per cent. of the patients. A negative Gmelin's reaction is no proof of the absence of bile, as the test is not a very delicate one. I believe that the presence of bile in the stomach contents after gastro-jejunostomy is almost, if not quite, the rule. My reason for this statement is the observation that almost invariably (in 99 per cent. of my cases) there is, after gastro-jejunostomy, an increase of the mineral chlorides in the gastric contents. What is the reason of this increase? Seeing that, as a rule, after gastro-jejunostomy (in 75 per cent. of my cases) there is a diminution of the total chlorides in the gastric contents, the increase in the mineral chlorides cannot be due to greater activity of the gastric mucous membrane. This increase of chlorides must be due to some other cause, and I think the inference is irresistible that the increase is due to the mineral chlorides of the bile and pancreatic juice which gain entrance to the stomach through the anastomosis. Evidence in favour of this view is afforded by studying the effect of undoing a gastro-jejunostomy on the amount of mineral chlorides in the gastric juice. On several occasions I have had an opportunity of performing gastric analyses on patients before and after gastro-jejunostomy and again after the gastro-jejunostomy has been undone. The result has always been the same, an increase in the mineral chlorides after gastro-jejunostomy, and a decrease towards the normal after the restoration of the alimentary canal to its normal condition. This is well shown in the following illustrative case :

Gastric Analysis

Before Gastro-jejunostomy—

Total Chlorides	0·420
Free HCl	0·051
Protein HCl	0·289
Mineral Chlorides	0·080

After Gastro-jejunostomy—

Total Chlorides	0·365
Free HCl	0·000
Protein HCl	0·153
Mineral Chlorides	0·211

After the Gastro-jejunostomy *was undone*—

Total Chlorides	0·343
Free HCl	0·018
Protein HCl	0·270
Mineral Chlorides	0·055

If my explanation of the increase of mineral chlorides be correct, then in those cases in which, in addition to gastro-jejunostomy, an entero-anastomosis is performed, we should not expect to find this increase, inasmuch as the bile and pancreatic juice are diverted through the entero-anastomotic opening. I have had an opportunity of investigating this point in three cases. In all of them there was, not an increase, but a decrease in the mineral chlorides, as shown in the following illustrative case :

Gastric Analysis

Before Gastro-jejunostomy—

Total Chlorides	0·335
Free HCl	0·003
Protein HCl	0·175
Mineral Chlorides	0·156

After Gastro-jejunostomy and Entero-anastomosis—

Total Chlorides	0·350
Free HCl	0·000
Protein HCl	0·226
Mineral Chlorides	0·124

In this case, notwithstanding the exceptional increase in the total chlorides, there was a decrease in the mineral chlorides.

One further point must be dealt with in this connection. Does this increase in the mineral chlorides occur after operations other than gastro-jejunostomy ? For example, take the operation of appendicectomy, is there an increase in the mineral chlorides after this operation ? In 26 per cent. of my cases of appendicectomy there was a marked decrease, in the remaining 74 per cent. there was an increase, but whereas after gastro-jejunostomy the increase in mineral chlorides is accompanied by a decrease in the total chlorides, after appendicectomy the increase in mineral chlorides is accompanied, as a rule, by an increase in the total chlorides.

I find that the average increase in mineral chlorides in the gastric contents, after gastro-jejunostomy, is 0·077 per cent. Doubtless part of this increase is due to the partial neutralisation of free hydrochloric acid and consequent formation of sodium chloride. This does not affect my argument, because (1) It must be the carbonates of the bile and pancreatic juice which cause this neutralisation, and (2) if free hydrochloric acid be absent from the gastric contents before gastro-jejunostomy, there is still an increase in the mineral chlorides after gastro jejunostomy. Roughly speaking, the bile and pancreatic juice contain 0·4 per cent. of mineral chlorides. I think, therefore, we may conclude that, on the average, the gastric contents after gastro-jejunostomy contain between 10 and 15 per cent. of bile and pancreatic juice. The average amount of bile, therefore, which regurgitates into the stomach is between 5 and 10 per cent.

Granted, then, that there is a permanent reflux of bile into the stomach after gastro-jejunostomy, the question arises, Is its presence harmful ? All the evidence indicates that it is not, provided the efferent opening of the anastomosis is efficient. Dastre and Masse have shown that in dogs the

presence of bile in the stomach does not interfere with the digestion and general health.   The well-known case reported by Sir Berkeley Moynihan, as well as the results of the operation of cholecysto-gastrostomy in man, confirm these observations, while the metabolism experiments, to which I will refer later, afford further evidence to the same effect.   Indeed, I think we may affirm safely that, in part at least, the beneficial effects of gastro-jejunostomy on gastric ulcer are due to the entrance of the alkaline pancreatic juice and bile into the stomach.

The Effect of Gastro-jejunostomy on the Gastric Secretion. —A most striking effect of gastro-jejunostomy on the gastric contents is the marked diminution of the total acidity. From examination of a large number of cases I find that the average diminution of the total acidity after gastro-jejunostomy is 30 per cent.   To what is the diminution of the total acidity due ?   Doubtless it is due, in part, to the neutralisation of the free hydrochloric acid by the bile and pancreatic juice which gain entrance into the stomach, but there are several facts which appear to me to indicate that this is not the only factor at work.   First, judging by the amount of bile usually found in the stomach after gastro-jejunostomy, the lowering of the total acidity is too great to be the result of a purely chemical reaction.   Secondly, if the whole of this diminution were due to neutralisation of the free hydrochloric acid by bile and pancreatic juice, it would mean that bile and pancreatic juice were gaining access to the stomach in such quantity that we should expect all the free hydrochloric acid in the gastric contents to be neutralised.   This is by no means so.   Of the cases in which free hydrochloric acid was present in the gastric contents before operation, in 48 per cent. free hydrochloric acid was still present after operation, although in diminished quantity. Thirdly, there is an almost constant diminution of the total

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chlorides in the gastric contents after gastro-jejunostomy. Now, seeing that there is, as I have already pointed out, an increase in the mineral chlorides, the diminution of the total chlorides must be attributed to diminished secretion. How is this diminished secretion to be explained ? The brilliant researches of Pawlow and his co-workers have demonstrated the striking interdependence of the secretions of the digestive glands, and it is this interdependence which seems to me to offer a reasonable explanation of the diminution of chlorides. I think we are justified in assuming that during the churning movements of the stomach, some of the gastric juice will be squeezed, as it were, through the anastomotic opening. This escape of some of the acid gastric contents will prevent the total acidity rising as high as otherwise would be the case. But it will have a further effect. We know from the researches of Wertheimer that the presence of an acid fluid in the jejunum causes a secretion of pancreatic juice. Therefore, after gastro-jejunostomy, if my hypothesis be correct, acid gastric contents will reach the jejunum at an earlier stage of digestion than would be the case in a normal alimentary canal. Consequently the flow of pancreatic juice will commence earlier, and there will be an earlier diminution of the gastric secretion, and as a consequence of this, a diminution in the total chlorides secreted by the gastric mucous membrane. Possibly other factors are at work, such as an inhibitory effect of secretin or other constituents of the pancreatic juice. In some cases hyperacidity may be due to spasmodic pyloric stenosis. This will be relieved by gastro-jejunostomy, and it is certainly significant that it is in the cases with hyperacidity that the diminution in the total acidity of the gastric contents is most marked.

The Effect of Gastro-jejunostomy on the Motility of the Stomach.—Diverse views have been expressed on this subject. Hartmann and Soupault, Rydygier and Rosenheim

found the motility of the stomach impaired permanently ; Carle and Fantino, on the other hand, maintain that the emptying of the stomach is hastened rather than retarded. I have investigated this point in thirty-two patients, and find that, as a rule, in cases in which there is no organic stenosis of the pylorus, the evacuation of the stomach is slightly accelerated. Usually the stomach is empty in from three to four hours after a meal. Another means of comparing the motility of the stomach before and after operation is afforded by the study of the amounts recovered one hour after a test meal. In 60 per cent. of a series of investigated cases the amount recovered after a test meal was less after operation than before, but the difference is not very great. In 66 cases the average amount recovered one hour after a test meal was 190 c.c. before operation, and 180 c.c. after operation. In all cases in which there is organic pyloric stenosis the motility of the stomach is improved by gastro-jejunostomy. I think, therefore, we may conclude, that in those cases in which the gastric motility is impaired markedly by pyloric stenosis or by adhesions, the operation of gastro-jejunostomy results usually in a marked improvement in the evacuation of the stomach contents. In those cases, on the other hand, in which before operation the motility is unimpaired, gastro-jejunostomy usually slightly hastens, but occasionally retards, the evacuation of the stomach, but inasmuch as this retardation or acceleration falls within physiological limits, we are justified in saying that in cases in which, before operation, the motility of the stomach is unimpaired, the evacuation of the stomach is unchanged by gastro-jejunostomy. I think that these observations on the motility of the stomach are against the view that gastro-jejunostomy is a drainage operation.

The Effect of Gastro-jejunostomy on Gastric Digestion.— —Some authorities maintain that gastric digestion is lost

after gastro-jejunostomy. Even if this were true, it is not of such importance as would appear at first sight, for we know from the experiments of Pachon and Carvalho on dogs, from the observations made on Schlatter's celebrated case of total gastrectomy, as well as from clinical evidence afforded by other patients who have survived complete removal of the stomach, that Nature is able to dispense with this organ. Personally, I do not believe that loss of gastric digestion is a necessary consequence of a short circuit between the stomach and the jejunum. Those who have expressed an opposite view base their conclusions on the subsequent diminution of chlorides found in the gastric contents.

This argument is not conclusive, as the chlorides may be diminished greatly without loss of gastric digestion. Further, unless at the time of operation atrophy of the gastric mucosa is already extensive, there is, in my experience, usually a progressive increase in the chlorides secreted during the digestion of a test meal. Again, if it be true, as stated by Schmidt, that only the gastric juice can digest raw connective tissue, by feeding a patient on raw beef and examining the fæces microscopically for connective tissue, we have a ready means of ascertaining the presence or absence of gastric digestion. I have carried out this test in fourteen patients, and in none of them did I find any undigested connective tissue on microscopical examination of the fæces. As estimated by the carmine red fibrin test, the degree of impairment of gastric digestion appears to be directly proportionate to the diminution of free hydrochloric acid. When, after gastro-jejunostomy, free hydrochloric acid is absent, there is considerable impairment of digestion, the nearer the amount of free hydrochloric acid approaches to the normal the better the digestion of fibrin.

The Effect of Gastro-jejunostomy on the Metabolism of the Human Body.—In the year 1897 Jöslin published some

observations on the metabolism of two patients on whom gastro-jejunostomy had been performed. He maintained that, as a result of this operation, there is a marked diminution of the proportion of nitrogen absorbed from the food, and an even more marked diminution in the fat absorption. These observations were accepted without question, and evil consequences were attributed to gastro-jejunostomy of which it is not guilty.

In my experience the average diminution of nitrogen absorption after gastro-jejunostomy is 1·7 per cent. and the average diminution of fat absorption 1·9 per cent. In all the cases I have investigated, the variation of absorption, both of nitrogen and of fat, has been within the limits found in individuals who are in good health. My observations have been repeated by Dr. H. C. Cameron, who has obtained very similar results.

These experimental observations are supported by the evidence of clinical experience. After gastro-jejunostomy patients may live for years in the enjoyment of perfect health. Even more striking are the results of gastro-jejunostomy for infantile pyloric stenosis. There are children alive and in robust health on whom this operation was performed in early infancy. It is inconceivable that they would have lived for seven or eight years, and developed into strong, healthy children, had the operation the harmful effects which have been attributed to it.

SUMMARY

To sum up.—We are justified, I think, in drawing the following conclusions as to the physiological effects of gastro-jejunostomy :

1. A certain amount of bile and pancreatic juice enters the stomach after gastro-jejunostomy, but the amount is

small, on an average between 10 and 15 per cent. of the gastric contents.

2. The total acidity of the gastric contents is diminished, on an average by 30 per cent. This is due partly to a diminution of the total chlorides secreted by the stomach, and partly to the partial or complete neutralisation of free hydrochloric acid by the alkaline bile and pancreatic juice which enter the stomach through the anastomosis, and probably also to earlier stimulation of the pancreatic secretion, and compensatory earlier lessening of the gastric secretion.

3. Gastric digestion is impaired, but not lost, after gastro-jejunostomy.

4. The motility of the stomach, if normal before operation, is for all practical purposes unaffected. This is against the view that gastro-jejunostomy is a drainage operation.

5. Gastro-jejunostomy has no material effect on the metabolism of the human body, the percentage of nitrogen and fat absorbed being within the limits observed in individuals who are apparently healthy. This chemico-pathological evidence is supported by the evidence of clinical experience.

I have thus stated briefly what I believe to be the effects of gastro-jejunostomy on the functions of the stomach. In conclusion, it remains to be considered what effects these changes have on a gastric or duodenal ulcer. In other words, how does a gastro-jejunostomy act ? Clearly, where there is a pyloric stenosis, the chief use of gastro-jejunostomy is the provision of a new outlet for the gastric contents. This function of gastro-jejunostomy does not require further discussion. It is with cases in which there is no organic stenosis that I am now dealing. I think it is clear that drainage does not explain the beneficial effects of gastro-jejunostomy on ulcers. From a study of my cases it is

evident that the relief afforded does not depend on hastened evacuation of the stomach. After gastro-jejunostomy the evacuation of the stomach may be markedly retarded, and the amount recovered after a test meal increased considerably, and yet the patient gains relief, and remains permanently well. This is, I think, conclusive evidence against the mechanical hypothesis as to the action of gastro-jejunostomy. To my mind the conclusion is irresistible that gastro-jejunostomy is a physiological operation.

It is easy to ascertain the changes in the gastric contents which follow gastro-jejunostomy, but at present we must admit that we can only speculate as to which of those changes is the important factor in the relief of the patient. As I have already pointed out, a most striking feature after gastro-jejunostomy is the diminution of the total acidity. As a rule there is also a diminution of the active hydrochloric acid. Even if the active hydrochloric acid be not diminished after gastro-jejunostomy or, as sometimes happens, be increased, there is still a diminution of the total acidity. This suggests that it is the lowering of the total acidity which is of importance, rather than merely the diminution of the active hydrochloric acid. Possibly organic acids are of more importance than is supposed, and the diminution of the organic acids is one of the factors at work. That diminution of the free hydrochloric acid is not the chief factor is clear, since before operation free hydrochloric acid may be absent entirely, and yet the patient gains relief from all symptoms. My impression is, that in patients who have no free hydrochloric acid before operation the results are not so good as in those who have had free hydrochloric acid. This is due probably to impairment of gastric digestion as the result of absence of free hydrochloric acid.

In cases in which there is severe chronic gastritis with abundant secretion of mucus, possibly a gastro-jejunostomy

opening acts as a less rigid sentinel than the pylorus, and so more readily permits the escape from the stomach of indigestible mucus.

Probably the lowering of the total acidity is but a part of the aid which surgery gives to Nature. The presence of bile and pancreatic juice in the gastric contents after gastro-jejunostomy is so constant, that it is to the action of these juices I would give pride of place in the therapeutical effects of gastro-jejunostomy. It would seem that they do something more than merely modify the acidity of the gastric contents. Haply they may produce an emulsion, or exercise some specific influence, as to the nature of which we are completely in the dark. Much of the workings in Nature's laboratory are beyond our ken.

What then are the practical lessons to be learned ? I believe that they are these :—First, occlusion of the pylorus is an unnecessary complication of gastro-jejunostomy, and is based on erroneous pathology. Secondly, if gastro-jejunostomy be a physiological operation, then it is as efficient a treatment for ulcers of the body of the stomach as for ulcers near the pylorus, in other words, gastro-jejunostomy is preferable to excision. Notwithstanding the admirable work on this subject which comes from the Mecca of Surgery—the Mayo Clinic—I regard as " not proven " the view as to the great frequency with which carcinoma is grafted on simple gastric ulcer. Granted, however, for the sake of argument, that it is proven, at any rate there is no evidence that such an event is frequent after a gastro-jejunostomy has been performed. My own experience coincides with that of Professor Kocher and Dr. Gressot, that malignant degeneration of ulcers occurs in less than 3 per cent. of all cases after the operation of gastro-jejunostomy for supposed simple ulcer. Viewed from this standpoint the teaching that excision of simple ulcers is advisable or necessary is

not based on established conclusions, and is contrary to clinical experience.

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CHAPTER VII

JEJUNAL AND GASTRO-JEJUNAL ULCERS

THE first case of jejunal ulcer was reported by Braun in 1899. In 1909 I published a short monograph on the subject, with a review of all cases recorded up till that date.

I pointed out that in a considerable proportion of the cases recorded as jejunal ulcer, the ulcer was situated at the anastomosis, and therefore was gastric as much as jejunal. In order that such ulcers should be distinguished clearly from jejunal ulcer proper, I suggested that they should be described as " gastro-jejunal ulcers." This classification has been adopted by most of those who have written since on the subject.

The characters of these two varieties of ulcer differ, and I believe the conditions determining their occurrence are dissimilar. Jejunal ulcers are the result of altered physiological conditions produced by operation ; gastro-jejunal ulcers probably are a direct consequence of operation ; their origin is due to faults in technique, and their persistence to hyper-acidity.

Frequency.—It is impossible to estimate with accuracy the proportion of cases in which gastro-jejunostomy is followed by jejunal ulcer. Mikulicz had two cases in 160 gastro-jejunostomies. Schostak has reported that in the Krönlein clinic one instance of jejunal ulcer occurred in 92 gastro-jejunostomies. Wickenhauser observed three jejunal ulcers in 115 gastro-jejunostomies, and Rotgans one in 49. Jejunal ulcer occurred in three of the 348 patients whose

after history I have been able to trace. Doubtless some cases have not been recorded, and probably more have not been recognised. Even so the total number would be markedly small in comparison with the enormous number of times the operation of gastro-jejunostomy has been performed. Now that attention has been directed to the subject, doubtless more cases would have been recorded were jejunal ulcer at all common. There can be little doubt that with modern technique it is now a less frequent complication. In the present state of our knowledge I think we are justified in estimating the probable risk of jejunal ulcer following gastro-jejunostomy at under two per cent.

Clinical Groups

Clinically, the cases of jejunal ulcer hitherto recorded are divisible into two groups :

Group I.—Cases in which perforation into the general peritoneal cavity occurs.

Group II.—Cases in which, owing to the formation of limiting adhesions, perforation does not result in the escape of bowel contents into the general peritoneal cavity.

This group includes two subdivisions :

(a) Cases in which the base of the ulcer becomes adherent to the abdominal parietes, so that perforation results in inflammatory exudation into the abdominal wall.

(b) Cases in which the base of the ulcer becomes adherent to and perforates into a hollow viscus, the colon in the cases so far recorded.

Symptoms.—The clinical picture presented by the cases in which perforation into the general peritoneal cavity occurs, differs materially from that in cases in which general peritonitis is prevented by the formation of protective adhesions.

Symptoms in Cases belonging to Group I.—In almost all the recorded cases belonging to this group, the patients, after gastro-jejunostomy, had lost all gastric symptoms and had been in good health until, suddenly, "like a bolt from the blue," they were seized with severe abdominal pain, heralding a perforative peritonitis, which in the untreated cases, speedily had a fatal ending.

Symptoms in Cases belonging to Group II.—Pain, varying in intensity, and in many of the cases of a severe character, is the prominent symptom of cases in this group. In some instances the pain was not sufficiently severe to prevent the patients from following, more or less, their usual vocations, for some years before they sought surgical relief; while in others the patients were reduced to an extreme degree of exhaustion. The pain does not appear to have any definite relation to the ingestion of food, although often it is aggravated by it.

An important sign of jejunal or gastro-jejunal ulcer is the existence of an induration or swelling in the abdominal wall. Usually such a swelling is present if an anterior gastro-jejunostomy has been performed. This swelling is the result of the adhesion of the ulcerated area to the anterior abdominal wall. Perforation of the base of the ulcer leads to an inflammatory exudation into the abdominal wall, and consequent induration, and in some instances to a very definite mass. Sometimes the skin over the swelling becomes ulcerated and destroyed with the formation of a jejunal fistula. As a rule, the situation of the swelling is in the left rectus muscle about the level of the umbilicus. Thus the presence of a swelling in the left rectus is highly suggestive, if indeed not diagnostic, of jejunal or gastro-jejunal ulcer, while the absence of any induration or swelling makes the diagnosis of this condition more difficult and less certain. When a posterior gastro-jejunostomy has been

performed, as a rule such a swelling cannot be felt. There is usually hyper-acidity of the gastric contents.

Diagnosis.—In the cases in which perforation into the general peritoneal cavity occurs, the signs and symptoms are similar to those of a perforated gastric ulcer. We cannot distinguish between the two conditions, but it appears that after gastro-jejunostomy a perforated jejunal ulcer is a more likely event than a perforated gastric ulcer.

When protective adhesions are present, the diagnosis of jejunal or gastro-jejunal ulcer must often be uncertain. But this much we can say : that when, after an interval of good health, a patient, who has had gastro-jejunostomy performed, begins to suffer constant pain, especially if the pain be accompanied by hyper-acidity or hyper-secretion, we should bear in mind the possibility of the existence of a jejunal or gastro-jejunal ulcer. When, in addition to pain, there is an induration or swelling of the abdominal wall in the neighbourhood of the stomach, the diagnosis is almost certain. We must remember, however, that a swelling or induration rarely is present in patients on whom the posterior operation has been performed. For practical purposes, we may say that every case of recrudescent pain of a constant character after gastro-jejunostomy, should be regarded as a case of potential jejunal or gastro-jejunal ulcer, and treated accordingly.

Pathology.—First, as to jejunal ulcer proper. This in appearance and clinical course bears a close resemblance to the ordinary round ulcer of the stomach and duodenum. As in the stomach, we have the " punched-out " ulcer suggestive of rapid destruction of tissue, apt to perforate before protective adhesions are formed. Naturally, the thin jejunal wall is destroyed more readily by ulceration than is the gastric wall, with its thicker musculature and tough submucous layer. Then, too, we have the jejunal ulcer (Fig. 48)

with shelving thickened edges, more chronic in course, be- .
coming adherent to neighbouring structures, finally per-
forating and leading to inflammatory infiltration of the
adherent structures. Jejunal ulcers are situated most com-
monly in the efferent limb, either opposite to or just below
the anastomosis, an observation which has a bearing on the
ætiology of these ulcers.

Secondly, as to gastro-jejunal ulcer (ulcer at the site of

JEJUNAL ULCER

Fig. 48.—A portion of resected jejunum, showing a chronic jejunal ulcer. From a
woman aged fifty-two years, on whom an anterior gastro-jejunostomy had
been performed five years previously.

anastomosis). Gastro-jejunal ulcer lacks the definite localised
appearance of the jejunal ulcer. It appears as an irregular
ulceration round the margin of the anastomotic opening,
sometimes invading the jejunal as well as the gastric mucosa.
If healing occur, the process of cicatrisation may lead to
partial, or even complete, obliteration of the communication
between the stomach and jejunum. Gastro-jejunal ulcers
are less likely to perforate into the general peritoneal cavity
than are jejunal ulcers.

Ætiology.—Various hypotheses have been adduced as
furnishing the explanation of jejunal ulceration following

gastro-jejunostomy ; and although, until our knowledge, as to the causation of gastric and duodenal ulcers, is more precise, conclusions regarding the ætiology of jejunal ulcers may require subsequent modification, nevertheless, we can form fairly reliable indications as to the conditions favouring its occurrence, and so come to some conclusions as to the best means of prevention and treatment. The three chief hypotheses which have been put forward to explain the occurrence of jejunal ulcers are as follows :

1. That jejunal ulceration is due to circulatory disturbances in the attached jejunum.

2. That jejunal ulcer is an infective process.

3. That jejunal ulcer is due to the digestive action of gastric juice on mucous membrane accustomed to the presence of alkaline contents only.

The " peptic " hypothesis has been adopted by most of those who have written on this subject, so that jejunal ulcer following gastro-jejunostomy usually is spoken of as " peptic " jejunal ulcer. This appears to me to be putting the cart before the horse ; the digestion of the living cells must be the consequence of their injury by the hydrochloric acid of the gastric juice. The peptic action is secondary, the primary factor being the injury of the mucous membrane by the toxic action of the hydrochloric acid. I believe that the amount of free hydrochloric acid, not the total acidity of the gastric contents, is the important factor. The free hydrochloric acid acts as a poison on the cells of the mucous membrane, so injuring them that they fall a ready prey to digestive action. In a normal individual, probably all the free hydrochloric acid of the gastric contents is neutralised before the chyme leaves the duodenum. Now after gastro-jejunostomy, the gastric contents pass directly into the jejunum. Two circumstances, however, prevent the diversion of the gastric contents from exercising an

injurious effect upon the jejunal mucous membrane. As I have pointed out elsewhere, after gastro-jejunostomy there is, first, a distinct diminution of the chlorides secreted by the stomach, and secondly, some regurgitation of bile and pancreatic juice into the stomach. In consequence of these changes the percentage of free hydrochloric acid in the gastric contents is diminished markedly, so that after a satisfactory gastro-jejunostomy the amount of free hydrochloric acid entering the jejunum is so small, that it is neutralised at once by the bile and pancreatic juice.

My view is that jejunal ulcer following gastro-jejunostomy is the result of a toxic agent or poison which so injures or kills the cells of the jejunal mucous membrane that they are readily digested by the intestinal juice. The toxic agent usually present is free hydrochloric acid, but possibly other toxic substances may be present, and either may increase the effect of the other. Thus, a small percentage of free hydrochloric acid in the jejunum, which by itself would not cause ulceration, may in the presence of some other toxic agent, produce ulceration. The circumstances under which free hydrochloric acid may be present in the jejunum are :

1. Hyper-acidity of the gastric contents, so that the bile and pancreatic juice are unable to neutralise completely all the acid entering the jejunum.

2. Normal percentage of hydrochloric acid in the gastric juice but excessive secretion, so that the amount of hydrochloric acid discharged into the jejunum is greater than can be neutralised.

3. Diversion of the course of the bile and pancreatic juice, so that part of the jejunum is exposed to the action of the gastric contents unmixed with bile and pancreatic juice as after operations of the " Y type," and gastro-jejunostomy with entero-anastomosis.

4. Normal acidity and normal amount of gastric secretion, but incomplete neutralisation in the jejunum, owing to temporary diminution of the flow of bile and of the secretion of pancreatic juice.

In a few cases jejunal ulcers are probably of infective origin. Such cases present the following features : (*a*) the ulcers occur within a very short period after gastro-jejunostomy ; (*b*) usually the ulcers are multiple.

So far I have discussed very briefly the ætiology of jejunal ulcer proper. Much of what has been said applies also to gastro-jejunal ulcer. Hyper-acidity is an important factor in both, but there is this difference : jejunal ulcers are a result of altered physiological conditions produced by operation ; probably gastro-jejunal ulcers are a direct consequence of operation. When a wound becomes ulcerated, the presumption is that the ulceration is a consequence of the wound. Beyond the general rule that jejunal ulcers occur most commonly in that part of the jejunum in which the acidity of the intestinal contents is greatest, we do not know what determines the site and area of ulceration. The site of a gastro-jejunal ulcer is determined by the wound made in effecting the anastomosis, and the ulceration persists and spreads if there be hyper-acidity of the gastric juice.

In connection with the origin of gastro-jejunal ulcer, the question arises, Does primary union take place between the mucous membranes of the stomach and jejunum when they are maintained in apposition by the use of an inner suture ? It is obvious that the necrosis produced by a Murphy's button must result in a ring of ulceration which heals by cicatrisation. Non-apposition of the gastric and jejunal mucous membranes, injury to the mucous membrane during the operation, or infection of the suture used for approximation must also result in healing by granulation. Drs. A. H.

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Gould and Harrington,[1] as the result of experiments on animals, conclude that an inner layer of stitches has little influence upon the healing of the wound. They found that whether the mucous membranes were apposed by suture or not, the mucous membrane round the margin of the anastomosis sloughed, and the separation of the slough left an ulcer which became covered over with mucous membrane in about three weeks. If this be true, then it follows that every gastro-jejunostomy is followed by a gastro-jejunal ulcer. If hyper-acidity or hyper-secretion exist, then the ulcer, instead of healing, may persist or spread, and eventually cause symptoms such as have been described in an earlier part of this chapter.

I must state my belief, that in human beings primary union of the gastric and jejunal mucous membrane may occur. To attain such a result rigid asepsis is imperative, an impossibility in operations on animals. In human beings, on the other hand, by adequate preparatory treatment and by careful dieting, it is possible to render sterile the upper part at least of the gastro-intestinal tract, and so to operate under conditions more favourable for primary union than can be obtained in experimental work on animals. I have had an opportunity of examining a considerable number of anastomoses from patients who have died at various intervals after gastro-jejunostomy. In all I found direct continuity between the gastric and jejunal mucous membrane, and, further, the thread used for suturing was encapsuled and had not been discharged into the intestine. In several instances microscopical preparations from recent anastomoses showed continuity of mucous membrane, and no evidence of

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[1] *The Technique of Operations on the Intestines and Stomach*, 1906, p. 20. The process of repair following the use of Murphy's button has been investigated by Barbat, and F. T. Murphy has described the process of healing after anastomosis by means of McGraw's elastic ligature. A. H. Gould, in the work to which I refer, gives an admirable summary of these researches.

the sloughing which Gould states is the invariable rule.
Whether primary union be possible or not, we should
employ the means most calculated to obtain it, and of this
at least I am convinced—that the use of an inner suture
ensures an opening which subsequently contracts less than
an anastomosis effected by a button, or by the use of serous
sutures only.

The regurgitation of bile and pancreatic juice which takes
place into the stomach after simple gastro-jejunostomy, must
be favourable to the union of the apposed surfaces, by
diminishing the acidity of the gastric contents as they pass
through the opening, and Nature in her wisdom has so
ordered it, that this regurgitation is at its maximum just
when it is most needed—in the early days after operation.
This protective regurgitation, so far as I have observed, is
absent in operations of the " Y type " and when an antero-
anastomosis is performed.

The Closure of Gastro-jejunostomy Openings. — A few
years ago we heard a good deal about the closure of gastro-
jejunostomy openings in cases of patent pylorus. The
Radiographers stated that after gastro-jejunostomy the food
continues to pass out through the patent pylorus, and it
was assumed that as the anastomotic opening becomes
functionless, it gradually contracts, or becomes completely
closed. Now it is obvious, that closure of a gastro-jejunos-
tomy opening premises ulceration. The closure is the conse-
quence of a gastro-jejunal ulcer.

A gastro-jejunal ulcer is the result of inaccurate apposi-
tion of the mucous membrane, or too tight constriction
thereof. The ulcer may heal speedily, or may extend and
result in marked contraction or obliteration. Now just as
hyper-acidity is the condition which prevents healing of a
gastric ulcer, so hyper-acidity plays the chief rôle in the
prevention of the healing of a gastro-jejunal ulcer. Closure

of the anastomotic opening occurs, not because the pylorus
is patent, but because the cases in which the pylorus is
patent usually are cases of ulcer with hyper-acidity. The
hyper-acidity is the causal factor, not the patent pylorus.
It is in such cases that gentle handling of the mucous mem-
brane, careful application of the sutures, rigid asepsis, and
strict dieting after operation are especially important, in
order to prevent the occurrence of gastro-jejunal ulcer
with its attendant risk of subsequent contraction of the
opening.

If jejunal ulcer be the result of the toxic action of hydro-
chloric acid on the jejunal mucous membrane, it follows that
operations of the " Y type " are wrong in principle. After
this type of operation the bile and pancreatic juice enter the
jejunum some inches below the anastomosis, so that this
and several inches of jejunal mucous membrane are exposed
to the action of unneutralised gastric contents. Mons. le
Dr. Monprofit, one of the few advocates of this operation at
the present time, has obtained good results from it ; never-
theless, I think that it is a method which, if employed at
all, should be used only in those cases in which free hydro-
chloric acid is absent from the gastric contents. Primary
entero-anastomosis is an unnecessary and now seldom per-
formed complication of gastro-jejunostomy, and is open to
the same objection as gastro-jejunostomy " En-Y." Inas-
much as the relative frequency with which these methods
have been employed is unknown, we cannot prove that they
are followed by jejunal or gastro-jejunal ulcer more fre-
quently than simple anterior or posterior gastro-jejunostomy.
But if, as is almost certainly the case, operations of the
" Y type " (including entero-anastomosis) have been far less
commonly performed than the simple anterior or posterior
method, it is perhaps not without significance that in 24
per cent. of the recorded cases, jejunal or gastro-jejunal

ulcer has followed gastro-jejunostomy " En-Y " or gastro-jejunostomy with entero-anastomosis.

A perusal of the recorded cases shows that a large proportion have followed the anterior operation, and it has been argued that this method predisposes to jejunal ulceration. At first sight this appears to be the case, but I do not think that a closer examination of the records justifies this assumption.

Very few instances of jejunal ulcer following a posterior, short-loop operation have been recorded. It has been suggested that the upper part of the jejunum is more resistant than the lower to the toxic action of hydrochloric acid. I think this is doubtful. Possibly the comparative immunity of the short-loop operation may be due, not to any difference in the physiology of this method, but to improvements in technique in gastric operations in general.

The Cause of Hyper-acidity after Gastro-jejunostomy.— I have already pointed out that after gastro-jejunostomy there is a marked fall in the total acidity of the gastric contents, the results of the altered physiological conditions effected by the operation. This diminution, as a rule, is sufficient to convert hyper-acidity before operation into hypo-acidity after operation. What, then, are the causes of hyper-acidity after operation ? A clear distinction must be drawn between two classes of case :

1. Cases in which hyper-acidity is present before and *persists* after operation.

2. Cases in which, after a period of normal or subnormal acidity following operation, the gastric contents subsequently become hyper-acid.

Hyper-acidity may persist after operation for the reason that before operation the hyper-acidity was so extreme, that the diminution caused by gastro-jejunostomy is insufficient to reduce the acidity of the gastric contents to normal,

unless the patient be kept on a milk diet for some months. Hyper-acidity may persist also because of a too small or inefficient or defective anastomotic opening.

Hyper-acidity of the gastric contents succeeding a period of normal or subnormal acidity is due probably to one of two causes; too small an opening, or indiscretions in diet.

In some cases both these factors may be present. When gastro-jejunostomy is performed on a dilated stomach, the operation usually is followed by a considerable diminution of the size of this organ, and so it may happen that an opening originally of good size may become too small owing to the contraction of the stomach around it. Hayem states that the presence of an acid liquid in a fasting stomach is an indication that the opening does not allow of complete evacuation of the stomach. A small or inefficient opening leads to gastric stasis, gastritis, and hyper-acidity. I quite admit that a slight degree of gastric stasis may have no ill result; but it is reasonable to argue that in such a case a slight attack of gastritis due to some error in diet, insufficient to cause trouble if the anastomotic opening were larger, may lead to such a degree of gastric stasis that hyper-acidity ensues. Doubtless in some cases long-continued indiscretions in diet may lead to gastritis, however large the opening may be.

Treatment.—The treatment of the cases in which perforation into the general peritoneal cavity occurs is obvious. Immediate laparotomy and suture of the perforation offer the only prospect of saving the life of the patient. If the patient recover, the condition of the gastric contents should be investigated, and if hyper-acidity, hyper-secretion, or gastric stasis exist, the gastro-jejunostomy opening should be enlarged, or a new gastro-jejunostomy performed.

In the cases of Group II in which protective adhesions are present, the indication for surgical treatment is not so

clear. The results of operation are not such as to encourage us to resort to surgery until after a trial of medical treatment.

In connection with medical treatment the question arises, What evidence have we of the spontaneous healing of jejunal and gastro-jejunal ulcers? In some of the recorded cases obliteration of the gastro-jejunostomy opening has occurred, and this obliteration must have been the result of cicatrisation following ulceration. It is true that in those cases surgical intervention was necessary subsequently, nevertheless they show that ulceration at the site of the anastomosis under some circumstances may heal spontaneously.

The evidence as to the possibility of permanent relief by medical treatment is very inconclusive. All we can say for certain is, that occasionally jejunal and gastro-jejunal ulcers heal, but the process of healing is apt to result in a condition requiring surgical intervention.

Medical Treatment.—In my judgment, before an operation is performed, the patient should undergo a course of careful dietetic and medicinal treatment for two reasons : first, there is just a possibility, remote it is true, that in this way a cure may be brought about ; and secondly, if subsequently operation be necessary, success will be more likely to follow if, as the result of medical treatment, the acidity of the gastric contents be diminished. The patient should be kept at absolute rest in bed, and on a diet of milk and eggs, as recommended by Lenhartz for the treatment of gastric ulcer. Bismuth, combined with small doses of hydrocyanic acid, usually relieves the pain, and the researches of Pawlow appear to me to show conclusively the value of the administration of alkalis in diminishing the acidity of gastric contents, an opinion confirmed by my experience in the cases I have seen.

If under medical treatment the pain disappear and the

acidity become diminished, we may persevere in the hope of ultimate cure, and the treatment should be prolonged for six months at least. The two indications for surgical treatment appear to me to be : first, persistence of the pain and hyper-acidity in spite of treatment, and secondly, evidence of hyper-secretion, or of gastric stasis. The presence of these conditions indicates that the anastomotic opening is at fault, and under these circumstances there is little likelihood of complete and permanent cure without surgical intervention.

Surgical Treatment.—The surgical treatment adopted must depend necessarily on the condition found at operation. Adhesions must be separated, and the perforation sutured, if there be one. A study of the recorded cases indicates plainly, that the less extensive the operation performed, the better is the result, especially if marked hyper-acidity exist. The anastomotic opening should be examined carefully, and if it be small it should be enlarged, or a new gastro-jejunostomy performed. In my judgment the former is the preferable course, as it is not only simpler, but does not necessitate a " Y type " operation, which, for reasons already given, is not to be recommended. If, on account of severe pain or rapid loss of strength, operation has to be undertaken before an attempt has been made to reduce any hyper-acidity which is present, I would suggest that the enlargement of the gastro-jejunostomy should be deferred ; but if, on the other hand, a strict course of medical treatment has been ineffectual in reducing acidity, then no object is gained by deferring the completion of whatever operative procedure is requisite.

In a number of cases, unfortunately too numerous, the symptoms have recurred after surgical treatment. In such it might be worth trying the effect of a gastro-jejunostomy or " En-Y," but with implantation of the proximal limb of

the jejunum into the stomach, so that the bile and pancreatic juice are diverted directly into the stomach.

Our great aim should be the prevention of jejunal or gastro-jejunal ulcer, and I will indicate very briefly, under two headings, what appear to me the points of importance in this connection :

(1) **The technique of gastro-jejunostomy.**

(2) **The after-treatment.**

(1) The Technique of Gastro-jejunostomy

First, I would emphasize the necessity for a large opening. There is invariably some contraction of the opening, and in cases of dilated stomach subsequent diminution in the size of this viscus still further narrows the communication between the jejunum and stomach.

Secondly, accurate apposition of the mucous membrane of the stomach and jejunum, so as to secure, if not primary union, at any rate, union with the formation of a minimum of scar tissue.

Thirdly, careful application of the inner suture so as to control bleeding, but, at the same time, so as to avoid such a degree of tightness as will cause localised necrosis of the tissues.

Fourthly, the use of simple suturing in preference to mechanical appliances, especially when hyper-acidity is present.

Fifthly, the avoidance of entero-anastomosis and of " Y-type " operations.

It has been suggested that catgut should be used for the inner suture in preference to silk or linen, on the ground that the latter is more apt to serve as a septic focus. I do not think there is a particle of evidence in favour of this assumption.

(2) The After-treatment of Gastro-jejunostomy

The necessity for prolonged after-treatment in cases of gastro-jejunostomy, perhaps has not received the attention which it deserves. My rule is to advise all patients whose gastric contents have been hyper-acid before gastro-jejunostomy, to avoid meat in any form for six months at least, and until such time as examination shows that the gastric acidity is sub-normal. The immediate relief which is experienced by patients on whom gastro-jejunostomy has been performed, tempts them to indulge in food unsuited to the condition of the gastric mucosa. In most cases in which gastro-jejunostomy is necessary, the mucous membrane is chronically inflamed, and many months must elapse before it is restored to a healthy condition.

SUMMARY OF CONCLUSIONS

1. The risk of jejunal ulcer following gastro-jejunostomy is probably under two per cent.

2. At the present time apparently this complication occurs less frequently than formerly.

3. Clinically, there are two groups of cases : (1) those in which perforation into the general peritoneal cavity ensues ; (2) those in which general peritonitis is prevented by the formation of adhesions.

4. Pathologically the cases may be classified as follows : (1) ulcers of the jejunum ; (2) gastro-jejunal ulcers, or ulcers at the site of the anastomosis.

5. In some instances jejunal ulcers are of infective origin ; in these cases ulceration commences within a very short interval after gastro-jejunostomy, and usually the ulcers are multiple.

6. In a large proportion of cases the ulcer is single, and is probably the result of the toxic action of hydrochloric acid,

which injures the cells of the mucous membrane so that they are digested by the intestinal juice. Possibly, other agents than hydrochloric acid may play a part in injuring the mucous membrane.

7. Gastro-jejunal ulcers are a direct consequence of the wound made in effecting the anastomosis, and their persistence is probably the result of hyper-acidity of the gastric juice.

8. Closure of a gastro-jejunostomy opening is the consequence of cicatrisation of a gastro-jejunal ulcer. It is more likely to occur when the pylorus is patent, not because of the patency of the pylorus, but because in such cases, hyper-acidity usually is marked.

9. Any procedure or disease which diminishes the amount of bile and pancreatic juice in the jejunum favours the occurrence of jejunal and gastro-jejunal ulcer. For this reason operations of the " Y type " and entero-anastomosis are inadvisable, at any rate in cases in which free hydrochloric acid is present in the gastric contents, as after these procedures the anastomosis and a portion of the jejunum are deprived of the protective influence of the alkaline bile and pancreatic juice.

10. The reason that ulceration has followed the anterior operation more frequently than the posterior operation with a loop, is probably that in former times the anterior operation was more frequently performed.

11. As so few instances of ulcer after the posterior short-loop operation have been recorded as yet, for the present we must assume that its occurrence after this type of operation is less likely. It is possible, however, that this immunity is partly the result of improvements in technique and in the after-treatment of gastric operations in general.

12. In cases in which perforation into the general peritoneal cavity occurs, immediate laparotomy offers the only chance of saving the patient's life.

13. Inasmuch as there is some evidence that jejunal and gastro-jejunal ulcers may heal, an operation should not be performed in the chronic cases, until after a thorough trial of medical treatment.

14. Even when surgical intervention is necessary an attempt should be made first to diminish hyper-acidity, if this be present.

15. Our aim should be to prevent the occurrence of this complication of gastro-jejunostomy. Preventive treatment consists in (1) careful and appropriate surgical technique, and (2) prolonged after-treatment.

16. Lastly, every case of recrudescent pain of a constant character after gastro-jejunostomy should be regarded as a case of potential ulcer, and treated accordingly, especially when the pain is associated with hyper-acidity or hyper-secretion.

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CHAPTER VIII

GASTRIC ULCER, ITS PATHOLOGY, SYMPTOMS, AND DIAGNOSIS

CLINICALLY, there are two varieties of gastric ulcer, the acute and the chronic. Whether these varieties are distinct pathologically is doubtful. Probably they are not. It is reasonable to suppose that the chronic ulcer originates as an acute ulcer, which, instead of healing, persists in the form we recognise as a chronic ulcer.

Pathologically the following varieties of gastric ulcer are described :

(i) Erosions

(a) **Simple erosions**, which appear as minute superficial abrasions of the gastric epithelium. During life these erosions are seen as numerous bleeding points, but after death they are difficult to detect.

(b) **Exulceratio simplex of Dieulafoy.**—This is simply a more extensive erosion with greater destruction of the surface epithelium. In some cases the blood-vessels of the muscularis mucosæ are exposed, and if ulcerated, very severe hæmorrhage may ensue.

(ii) Simple Ulcer

(a) The **acute round ulcer,** much written of but seldom seen. Its edges are sharply defined and clear-cut, giving it a punched-out appearance. As a rule, more of the gastric mucosa is destroyed than of the deeper layers, so that the ulcer has the shape of a funnel or truncated cone with its

PLATE XXXVI.
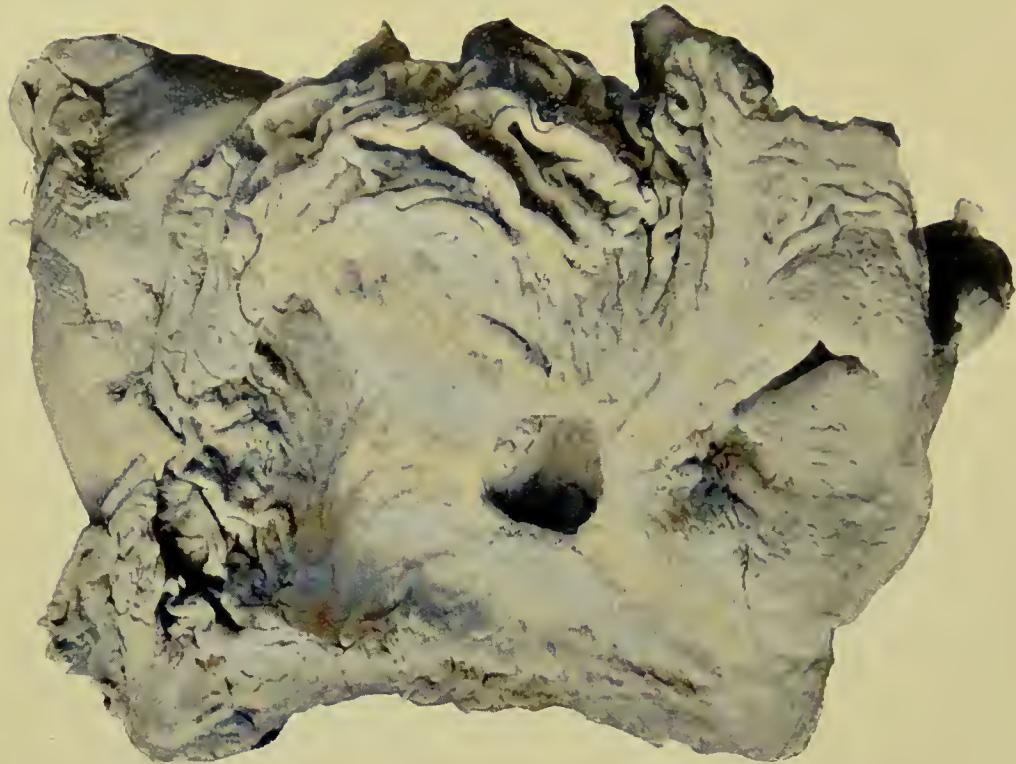
FIG. 49.—Chronic ulcer of quadrilateral shape on the posterior wall of the stomach, which formed a deep excavation on the substance of the pancreas. (W. Soltau Fenwick.)
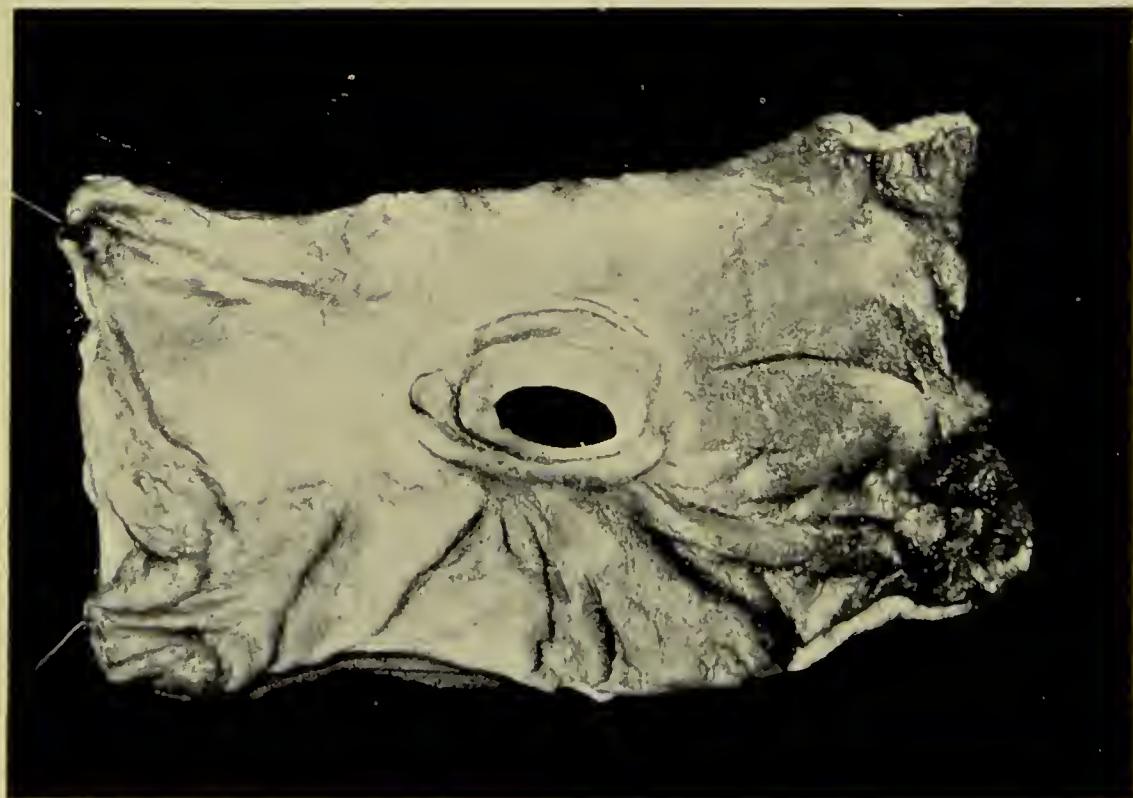
FIG. 50.—A chronic ulcer. The "terraced" appearance of the ulcer is well shown. (St. Bartholomew's Hospital Museum, No. 1911.)

base at the mucous surface and its apex towards the peritoneal surface of the stomach. The edges of the ulcer are not indurated, but there is often considerable œdematous swelling of the surrounding gastric wall.

(b) **Chronic Ulcer.**—The commonest variety of gastric ulcer, and the one which requires surgical treatment most frequently. Chronic ulcers vary greatly in size and shape. Usually they are oval or irregular rather than round, the edges being much indurated and raised above the level of the surrounding mucosa. The mucous membrane round the ulcer is deeply congested, numerous varicose veins often being visible. Usually the floor of the ulcer is composed of the muscular wall of the stomach, which is exposed by the destruction of the mucous membrane. In other instances, the whole of the gastric wall may be destroyed, so that the floor of the ulcer is formed by some neighbouring organ, such as the pancreas, to which the stomach, at the seat of ulceration, has become adherent before complete destruction of the whole thickness of the gastric wall has ensued.

Formerly, it was believed that gastric ulcer occurs far more frequently in women than in men. Since surgical activity has invaded the field of gastric disease, our ideas as to the sex-incidence of ulcer have been modified considerably.

We know now that symptoms, supposed to be pathognomonic of gastric ulcer, may arise from other abdominal diseases, such as chronic appendicitis, or gall-stones.

Formerly it was taught that sixty per cent. of gastric and duodenal ulcers occurred in women. According to Drs. W. J. and C. H. Mayo sixty-two men are affected to thirty-two women.

As, however, duodenal ulcer is much more frequent in the male, the incidence of true gastric ulcer in the two sexes is nearly equal, fifty-two men to forty-eight women.

Ætiology.—Many attempts have been made to ascertain the cause of gastric ulcers. The problem is not yet solved. Their origin is still a matter for speculation. Personally, I believe that both gastric and duodenal ulcers are toxic in origin. The occurrence of acute duodenal ulcers in some cases of severe burns, and also occasionally in cases of acute appendicitis, the association of appendicular disease with duodenal ulcer and with chronic gastritis, seem to point strongly to a toxic origin. In recent years some interesting experimental observations have been made by Dr. C. Bolton, which strongly support this view.

The Clinical History.—Sometimes it is possible to make a diagnosis of gastric ulcer from the history alone, therefore great care must be taken to obtain a complete and accurate record. This is often difficult, and requires much patience and careful interrogation.

The patient may state that the symptoms appeared quite suddenly, but cross-examination usually will elicit the fact, that for years before the sudden onset he had suffered from indigestion, to which little attention has been paid. The periodicity of the attacks of pain is a notable feature of gastric ulcer.

The attacks come on suddenly, and continue for days or weeks, and then cease abruptly. The patient may be free from symptoms for weeks, months, or, in exceptional cases, for years ; may consider himself cured until he is disillusioned by the onset of another attack. Gradually the intervals between the attacks become shorter and shorter, until pyloric stenosis, hour-glass stomach, or chronic gastritis ensue ; the symptoms become continuous and the patient a constant invalid.

SYMPTOMS

The three cardinal symptoms of gastric ulcer are : pain, hæmorrhage, and vomiting.

Pain.—This is by far the most important, most constant, and most characteristic symptom. It varies from slight discomfort, sometimes a little more than a feeling of fullness, to pain of such intensity that the patient is " doubled up."

Usually, it is referred to the epigastrium, and is limited to an area of from two to three inches in extent. Sometimes, in cases of ulcer towards the cardiac end of the stomach, the patient is very definite in localising the pain to the left of the middle line. As a general rule, the nearer the ulcer to the pyloric end, the more often is the pain referred to the right of the middle line ; but the site of the pain is not an infallible guide to the situation of the ulcer.

Radiation of the pain to the back is not so common as text-books would have us believe, but sometimes the pain radiates to the shoulders.

The pain comes on from half an hour to one hour after food : the nearer the ulcer is situated to the pyloric end of the stomach, the longer the advent of pain is delayed. In gastric ulcers close to the pylorus, the pain may not come on for three or four hours, and in such cases the differentiation from duodenal ulcers is difficult. (See illustrative case, p. 149.)

The pain increases in intensity as digestion progresses, and is at its maximum before the time of the next meal, so to the patient it seems as if the pain were the result of want of food. Sometimes taking more food relieves the pain, but this is not the case so often as in duodenal ulcer. Relief is obtained also by vomiting, whether spontaneous or artificially induced.

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It is common for those suffering from gastric or duodenal ulcer to state that the pain wakes them up in the early morning hours, and they find by experience that it is advisable to keep a glass of milk by the bedside, so that on waking they can drink it, and so secure temporary relief.

Almost all sufferers from gastric ulcer are troubled with flatulent distension of the stomach ; the gas is belched up, often with small quantities of fluid, which is very bitter or acid to the taste (water-brash).

Hæmorrhage.—Hæmorrhage from gastric ulcer is not so frequent as is supposed. Hæmatemesis is not due necessarily to gastric ulcer, and so it is not certain evidence of ulcer, neither does its absence contra-indicate a diagnosis of gastric ulcer. The amount lost may be so small that repeated examinations of the fæces are necessary to detect its presence (occult blood), or may be so great that it proves fatal immediately.

If vomited soon after the hæmorrhage has occurred, the blood is bright red in colour, and clots may be present. If the blood has been retained in the stomach for any length of time, the vomited material may be dark red, or a dirty brown colour (the so-called " coffee ground " vomit). If blood in considerable quantity pass through the pylorus, it gives rise to black stools (melæna).

Vomiting.—In the early stage of gastric ulcer, vomiting is infrequent. It may occur if the patient over-indulge in food, but as a rule a patient with a gastric ulcer learns by experience to avoid this indiscretion. When it occurs, vomiting comes on usually some hours after food, when the pain is at its worst.

Some patients produce vomiting artificially, in order to empty the stomach and so gain relief from the pain. Some years ago I operated on a lady who, in order to conceal the severity of her symptoms from her husband, took an ordinary

PLATE XXXVII.
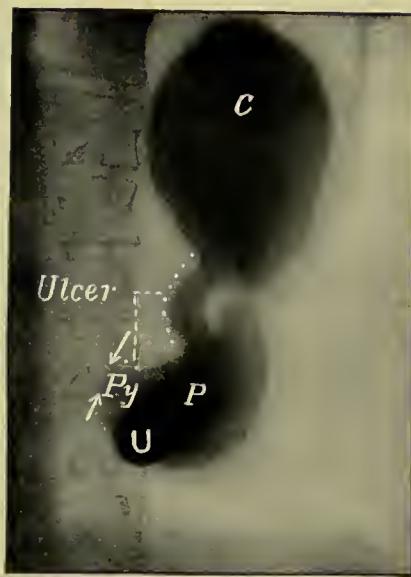
FIG. 51.—Taken on the couch after a bismuth meal, showing a typical chronic ulcer of the lesser curvature in a man, aged 28. There was pain half an hour after food, and tenderness to pressure under the left costal margin. There had been two attacks of hæmatemesis, one four, the other two years ago. In addition to the typical appearances of a chronic ulcer of the lesser curvature there was tight organic stenosis at the pylorus, with strong anti-peristaltic contractions occurring in groups, alternating with groups of strong waves in the normal direction. At the operation two chronic gastric ulcers were found: one at the pylorus, the other on the lesser curvature. There was scarcely any hour-glass constriction under the general anæsthetic. *C., P.,* cardiac and pyloric portions of the stomach; *Py.,* pylorus. The arrows indicate the pyloric stenosis.—By Dr. A. C. JORDAN.

dinner every night and immediately afterwards made herself vomit, to prevent the onset of the pain which she knew would follow invariably within an hour or so.

Vomiting in gastric ulcer may be due to one of three causes :

(i) In the early stages, vomiting of partially digested food, without much mucus, is reflex—Nature's effort to get rid of the irritating food.

(ii) In the later stages vomiting of small quantities of ill-digested food, with much mucus, is the result of chronic gastritis.

(iii) Vomiting of large quantities of sour fluid with partially digested food is an indication of pyloric stenosis, or hour-glass stomach.

Physical Examination.—As a rule there is tenderness over the epigastrium, but this is not constant. Between the attacks physical examination may reveal nothing abnormal. Sometimes the induration and adhesions round the ulcer form a mass sufficiently large to be felt on palpation. This is very important, as the presence of a swelling may lead to a diagnosis of cancer. In many such instances, patients have been denied operation which would have given them complete relief.

Examination of the Gastric Contents.—Hyper-secretion is not an invariable accompaniment of gastric ulcer. In more than half the cases the stomach is empty in the early morning. In 40 per cent. of the cases from 20 to 50 c.c. of clear acid fluid may be withdrawn by aspiration in the early morning. Unless the ulcer be near to the pylorus, or cicatrisation has resulted in pyloric stenosis or an " hour-glass " constriction, the motility of the stomach is unimpaired ; food-remains are not present in the morning, and the amount recovered after a test-meal is not greater than in health. When there is pyloric stenosis, food-remains

are present in the morning, and the amount of gastric contents recovered on withdrawing the test-meal may be very considerable.

When a gastric ulcer is situated near to the pylorus, as a rule, the free hydrochloric acid is increased ; on the other hand, when the ulcer is in the middle or at the cardiac end of the stomach, the free hydrochloric acid is diminished.   In cases of hour-glass stomach free hydrochloric acid is absent.

Probably the free hydrochloric acid always is increased in the early stage of the disease.   Later, there is a progressive diminution, so that when marked pyloric stenosis is present, free hydrochloric acid may be absent entirely.

The following are two typical analyses :

1. Gastric ulcer near the pylorus :

Total acidity	.	.	.	.	.	70
Total chlorides	.	.	.	.	.	0·419
Free HCl	.	.	.	.	.	0·040
Protein HCl	.	.	.	.	.	0·222
Mineral chlorides	.	.	.	.	0·156

2. Gastric ulcer at the cardiac end of the stomach :

Total acidity	.	.	.	.	.	84
Total chlorides	.	.	.	.	.	0·361
Free HCl	.	.	.	.	.	0·018
Protein HCl	.	.	.	.	.	0·244
Mineral chlorides	.	.	.	.	0·098

When a gastric ulcer is near the pylorus the clinical differentiation from duodenal ulcer often is impossible.

The following is a good illustrative case of gastric ulcer :

A. W., a hospital nurse aged thirty.   Patient has suffered from indigestion for many years. She dates her present illness to seven years ago, when she began to have attacks of severe pain and a feeling of fullness after meals. The onset of pain varied between ten minutes and three hours after food.   Until a year ago the attacks occurred at irregular intervals, and sometimes were accompanied by vomiting.

She has never had hæmatemesis. For the last year the attacks have come on regularly two hours after every meal, and almost invariably have been accompanied by vomiting. Patient has lost flesh considerably and is anæmic. Diagnosis: gastric ulcer. (See Analysis No. 2 on p. 148.)

At operation a large ulcer was found near the cardiac end of the lesser curvature, causing slight constriction of the stomach. An anterior gastro-jejunostomy was performed, and the patient was completely relieved of all her symptoms.

The following case illustrates the difficulty of differentiating gastric ulcer, near the pylorus, from duodenal ulcer, as the symptoms suggested duodenal rather than gastric ulcer.

H. B., aged forty-nine. For the last thirty years patient has suffered more or less from indigestion, water-brash, occasional vomiting, flatulence, and pain, coming on three or four hours after food. He usually has three or four attacks in the course of a year, and is comparatively well in the intervals. During the past three years, the attacks have been much more severe than formerly. He has never had hæmatemesis or melæna. The pain comes on three or four hours after food, and is relieved by water-brash, or by vomiting. Usually he is wakened up at night every four hours by pain or flatulence. For the last six months he has been getting steadily worse, and has lost nearly two stone in weight. He is now extremely emaciated. (See Analysis No. 1 on p. 148.)

At the operation a large indurated ulcer was found in the lesser curvature, about two inches from the pylorus. A posterior gastro-jejunostomy was performed, and the patient is now in excellent health, and has put on a stone and a half in weight.

Occasionally hæmorrhage is the most prominent symptom of gastric ulcer.

A lady, sent to me by Dr. Soltau Fenwick, had a severe hæmatemesis seven months before I saw her. She brought

up about a pint of blood, and her hæmoglobin was reduced to 21 per cent. A month later she had another, slighter hæmatemesis. Shortly afterwards she began to have pain in the stomach, which did not have any constant relation to food. A month later she had another attack of hæmatemesis and melæna, preceded by ten days of severe indigestion. Latterly, the pain had wakened her up at night. The patient was extremely anæmic.

Analysis of gastric contents :

Recovered one hour after test-meal—90 c.c. Good motility.

Total acidity	75
Total chlorides	0·325
Free HCl	0·040
Protein HCl	0·205
Mineral chlorides	0·080

Diagnosis : gastric ulcer.

On opening the abdomen I found a puckered ulcer in the stomach near the pylorus, and performed a posterior gastro-jejunostomy. The patient was completely relieved.

Diagnosis.—It must be admitted that often the diagnosis of gastric ulcer is very difficult. Many cases of supposed gastric ulcer are really instances of appendicular gastralgia. The symptoms are gastric, but the lesion is in the appendix. In other cases the symptoms are due to chronic gastritis, or to gall-stones.

When a gastric ulcer is close to the pylorus, differentiation from duodenal ulcer may be impracticable, as in the illustrative case given above (p. 149).

The points in the diagnosis are :

(i) The chronicity of the disease.

(ii) The periodicity of the disease.

(iii) The time relation of the pain to food, usually *one hour* after food, but often less than this period.

(iv) The diminution of free hydrochloric acid.

(v) The control of the pain and other symptoms by the administration of alkalis.

Differential Diagnosis.—Gastric ulcer has to be distinguished from duodenal ulcer, gall-stones, appendicular gastralgia, syphilis of the stomach, Bright's disease, gastroptosis, and intestinal stasis.

As pointed out already, a gastric ulcer near the pylorus may be indistinguishable clinically from a **duodenal ulcer.** As a rule, in gastric ulcers near the pylorus the onset of pain is from one to two hours, and in duodenal ulcer from two to three hours, or even longer, after food. Occasionally, however, in gastric ulcer, the interval between the taking of food and the onset of pain is several hours. The pain in duodenal ulcer is referred to the middle line, sometimes to the right of it. In gastric ulcer the pain is usually in the middle line, but not infrequently to the left of it. If there be radiation of the pain, it is usually to the right in duodenal ulcer, to the left in gastric, unless the ulcer be near the pylorus, when the radiation may be to the right.

Hæmorrhage is more often evidenced by hæmatemesis in gastric ulcer, and by melæna in duodenal ulcer.

In gastric ulcer, hyper-acidity, if present, is not marked ; more often there is hypo-acidity. In duodenal ulcer hyper-acidity is much more common than hypo-acidity.

In gastric ulcer hyper-chlorhydria is exceptional, unless the ulcer be near the pylorus. In duodenal ulcer hyper-chlorhydria is the rule, hypo-chlorhydria the exception. The hyper-tonic stomach so common in duodenal ulcer rarely is seen in gastric ulcer. Hyper-secretion is more common in duodenal than in gastric ulcer.

From **gall-stones** in the early stage, gastric ulcer often is hard to differentiate. Usually, close attention to the history will determine the diagnosis. In gall-stones the attacks of

pain or discomfort after food are slight, occur at irregular times, sometimes after food, sometimes not, are accompanied by much flatulence, often by a " catchy " pain on inspiration. The attacks pass off as suddenly as they have come, and are uninfluenced by drugs, or by diet.

In the later stage of gall-stones, the attacks of pain are more severe, and, like the milder attacks, come and go suddenly, bear no relation to food, radiate through to the back, and are sometimes of an agonising intensity never seen in cases of simple gastric ulcer. In gall-stone disease the gastric contents vary little from the normal, any change being in the decrease of hydrochloric acid and lowering of the total acidity. Hyper-secretion is sometimes present, but it is not common.

Chronic **disease of the appendix** frequently gives rise to gastric symptoms **(appendicular gastralgia)**. The pain is more indefinite, discomfort rather than pain, referred to the epigastrium, but less definitely to any particular spot. It follows quickly on the ingestion of food, and the kind of food makes little difference. Even milk causes discomfort. Sometimes the pain radiates to the right iliac fossa. Exercise and physical exertion bring on the pain in a manner that is rare in gastric ulcer. The symptoms are very chronic, often last for years with little tendency to get worse. In gastric ulcer, if untreated, usually there is progressive aggravation of the distressing symptoms.

The pain of appendicular gastralgia is uninfluenced by alkalis. Occasionally there is some tenderness over the appendix region. The gastric findings are those of chronic gastritis, much mucus, bad motility, diminished chlorides, absence or great diminution of free hydrochloric acid, and, as a rule, hyper-acidity, partly due to the increase of volatile acids. Commonly, there is hyper-secretion, and delayed evacuation of the stomach. (See also Chap. XXII).

The differentiation of a simple ulcer from **syphilis of the**

stomach is difficult, and at the same time of the utmost importance. If syphilis be the cause of the symptoms, the use of anti-syphilitic remedies is imperative, if the patient is to be relieved. Pain after food, and vomiting, are symptoms common to both complaints. If there be a history or evidence of syphilis, the possibility that the gastric symptoms are due to this cause must be considered.

In syphilis of the stomach hæmatemesis is not common, but there is often profound anæmia. The cachectic appearance of the patient is suggestive. The extreme severity of the pain, and its obstinacy to ordinary treatment, are the chief clinical features of gastric syphilis. Gastric analysis shows an extreme degree of chronic gastritis, great diminution of chlorides, low total acidity, and absence of free hydrochloric acid. In cases of doubt a Wasserman's test should be made.

In **Bright's disease,** digestive disturbances with anæmia and emaciation may suggest ulcer. The less severe character of the pain, the presence of albumen in the urine, the condition of the heart and vessels, usually enable a correct diagnosis to be made.

Gastric ulcer should not be confounded with **gastroptosis.** In gastroptosis usually there is hyper-acidity and normal or slightly diminished free hydrochloric acid. Discomfort rather than pain is the chief complaint. Contrary to what obtains in gastric ulcer, the patient usually is more comfortable on a solid diet. Distension of the stomach with air reveals the displacement of the viscus.

Many patients supposed to be suffering from gastric ulcer are in reality suffering from the effects of some form of **intestinal stasis.** Pain or discomfort, and tenderness over the stomach, nausea or vomiting, together with the pallid emaciated appearance of the patient, present a clinical picture suggesting gastric ulcer, but on exploration no ulcer is found. Not infrequently gastro-jejunostomy has been

performed in these cases, and naturally has not been followed by relief.

Intestinal stasis may be due to chronic disease of the appendix, or to bands at the lower end of the ileum, or in the cæcum or sigmoid, which cause definite mechanical difficulty in the onward passage of the intestinal contents. Occasionally there is a kink at the duodeno-jejunal junction (Lane) which interferes with the emptying of the duodenum.
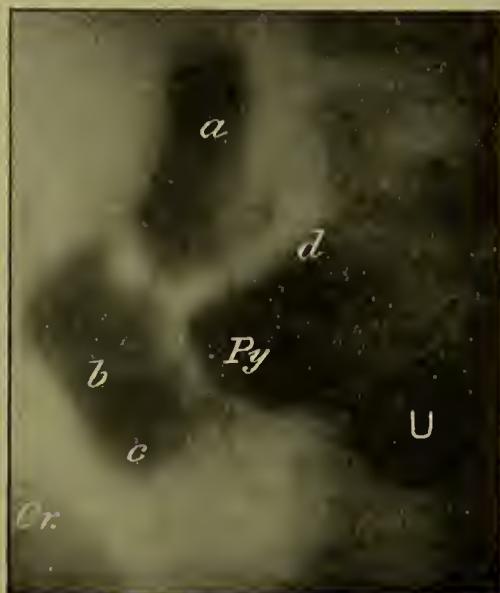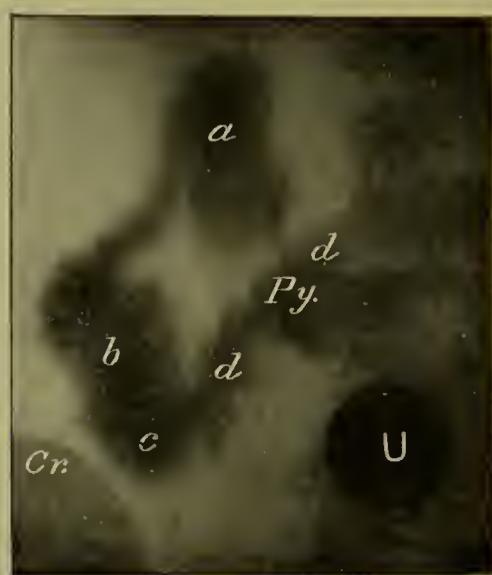
FIG. 52.   FIG. 53.

Figs. 52 and 53.—Pylorus and duodenum in a woman aged 33, a typical case of intestinal stasis, taken on the couch after bismuth, showing two phases of the strong "writhing" peristalsis by which the duodenum tried (in vain) to overcome the kink at the duodeno-jejunal junction. The bismuth was returned over and over again from the third to the second part of the duodenum—and even to its first part as each wave passed off. There was marked stasis in the ileum and in the large intestine. By Dr. A. C. Jordan. Py., pylorus. *a*, *b*, *c*, *d*, the four parts of the duodenum. U, umbilicus, marked by a penny. Cr. crest of right ilium.

The signs of intestinal stasis have been described admirably by Sir Arbuthnot Lane.

The main indications of intestinal stasis are :

1. The stained and inelastic condition of the skin.

2. The feebleness of the circulation, and the lowering of the blood-pressure.

3. The subnormal temperature.

4. The drowsy and apathetic condition of the patient.

5. The delay in the passage of fæces through the large bowel, as demonstrated by X-rays, or by the administration of charcoal.

In intestinal stasis, symptoms of auto-intoxication are marked, in gastric ulcer absent. In intestinal stasis, as a rule, the cæcum is markedly distended, and gurgling is felt readily. Tenderness is not limited to the epigastrium, but is present in both iliac fossæ, and often all over the abdomen.

In intestinal stasis, the symptoms usually are relieved at once by rest apart from diet ; in gastric ulcer, it is the diet, not the rest, which influences the pain.

In intestinal stasis, there is marked diminution of all the constituents of the gastric juice.

In intestinal stasis, the gastric symptoms are, I believe, due less to mechanical obstruction to the outflow of the gastric contents by back pressure, than to direct interference with the gastric secretions as the result of toxæmic absorption from the intestine. In support of this view, I may point out that when free hydrochloric acid is absent from the gastric contents in cases of intestinal stasis, before operation, after operation there is, not infrequently, an immediate restoration of the free hydrochloric acid in the gastric contents.

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CHAPTER IX

DUODENAL ULCER, ITS PATHOLOGY, SYMPTOMS, AND DIAGNOSIS

DUODENAL ulcer occurs in two forms, the acute and the chronic. Usually the acute ulcer is secondary to extensive burns of the skin, more rarely to acute appendicitis. It occurs also as a complication of septicæmia, pyæmia, or acute nephritis, and in the course of the specific fevers. Probably such ulcers are toxic in origin ; as a rule they cause no symptoms, and are recognised only on post-mortem examination. In other cases hæmorrhage or perforation ensues and hastens the death of the patient. When these complications result, the patients are already so seriously ill, that operation is not likely to prove of service. Therefore acute duodenal ulcer is of little surgical importance.

Chronic Ulcer of the Duodenum is by far the commoner and more important variety. It is situated most commonly on the anterior wall of the duodenum within three-quarters of an inch of the pylorus. Sometimes the ulcer extends up to, and is continuous with, the pylorus. Dr. W. J. Mayo was the first to call attention to the importance of the " pyloric vein " as a surgical landmark. By observing the relation of the ulcer to this vein, whether the ulcer is gastric or duodenal, may be determined without difficulty.

On the operation-table the site of the ulcer is indicated by injection or mottling of the peritoneal coat. In ulcers of long standing, the base may appear as a yellowish-white area with a depressed and puckered centre. On palpation the ulcer feels hard, like a ring of cartilage. If the wall of

PLATE XXXVIII.

FIG. 54.—A large duodenal ulcer, the base of which is formed by liver.   (Museum of the Royal Infirmary, Glasgow.)

the bowel be pushed in front of the examining finger the
" crateriform " shape of the ulcer often can be recognised
clearly. In the majority of cases there are no adhesions, but
in other instances the base of the ulcer may be adherent to
the liver, gall-bladder, or to a tag of omentum—Nature's
protection against acute perforation.

Cicatrisation of the ulcer may produce considerable
narrowing of the duodenal lumen. Sometimes the stricture
takes an annular form, dividing the first portion of the
duodenum into two compartments (" hour-glass duodenum ").
Dr. W. J. Mayo and Sir Berkeley Moynihan have recorded
instances of double stenosis (" trifid duodenum ").

How long a duodenal ulcer may exist before its presence
can be detected by inspection and palpation on the opera-
tion table, we do not know. It is possible, indeed probable,
that in the early stages an ulcer may be present which
cannot be felt. Some years ago I had an interesting case
bearing on this point. A lady, aged forty-two, had had
severe vomiting, hæmatemesis, and melæna. On opening the
abdomen, I found an enormously large appendix measuring
$3\frac{1}{2}$ inches in diameter, and containing three large, foul-
smelling concretions. I explored the stomach and duodenum
carefully, but could find no evidence of gastric or duodenal
ulcer. The symptoms recurred, and she had further severe
attacks of hæmatemesis and melæna. A year later I re-
opened the abdomen, and on this occasion found a well-
marked indurated duodenal ulcer adherent to the liver. I
have little doubt that, in this case, a superficial ulcer, which
could not be felt, existed at the time of the first operation.
The ulcer persisted, and in due time became indurated, so
that at the second operation it was palpable as well as recog-
nisable by sight.

More than one ulcer is present in about 15 per cent. of
the cases. When two ulcers exist, usually they are opposite

each other, suggesting that the second ulcer is due to infection from the first. Such ulcers have been termed " contact ulcers."

Formerly, it was believed that gastric ulcers were far more common than duodenal, the relative frequency being given variously as from five to ten gastric to one duodenal.

Many ulcers which were really duodenal were classed as pyloric and therefore gastric. Now that we can distinguish between gastric and duodenal ulcers, by observing the situation of the ulcer in relation to the pyloric vein, we know that duodenal ulcer is as frequent, if indeed not more frequent, than gastric ulcer.

In 200 patients operated on at the Mayo clinic, 60 ulcers (31 per cent.) were situated in the stomach and 119 (61 per cent.) in the duodenum.

In my own series of patients, in 60 per cent. the ulcer was in the duodenum, in 34 per cent. in the stomach, while in 6 per cent. there was an ulcer both in the stomach and in the duodenum.

As to the sex incidence : at the same clinic, duodenal ulcer was found 77 times in men and 23 times in women. Sir Berkeley Moynihan gives 73·6 per cent. in men, and 26·4 per cent. in women. In my own series, 80 per cent. of the duodenal ulcers have been in men and 20 per cent. in women. It is interesting to note that the sex incidence of cholelithiasis is just the opposite of this, namely 76 per cent. in women to 24 per cent. in men.

Ætiology.—The cause of duodenal ulcer is not ascertained as yet. Sir Berkeley Moynihan, in his classical work on duodenal ulcer, does not even discuss the ætiology.

In operating for duodenal ulcer I have been struck with the frequency with which there is coexisting appendicular disease. I have found that obvious disease of the appendix is present in 66 per cent. of the cases in which, at operation,

a duodenal ulcer was found. This association seems to me to be so frequent that it is not a mere coincidence, and some years ago I suggested elsewhere, that latent appendicitis is one cause of duodenal ulcer. The evidence of this is at present only presumptive. It is highly probable, in some instances, that gastric and duodenal ulcers are toxic in origin, and I think it is possible that duodenal ulcer is the result of toxic absorption from the appendix.

It is now established clearly, that appendicular disease may produce definite gastric symptoms, a condition for which I have suggested the term " appendicular gastralgia." Some years ago I pointed out that in these cases the gastric mucosa often shows an extreme degree of erosion. It is possible that this condition is the prelude to definite ulceration of the stomach or duodenum, the sequence of events being : appendicular disease ; secondarily, erosion of the gastric mucosa ; then superficial ulceration (the so-called " medical ulcer ") ; and later a demonstrable indurated ulcer. The brilliant work of Sir Arbuthnot Lane has thrown new light on obscure abdominal symptoms, and it is probable that intestinal stasis is an important factor in the ætiology of duodenal ulcer.

Clinical History.—In chronic duodenal ulcer three features are very definite and constant : the chronicity of the disease, the periodicity of the attacks, and the well-being of the patient in the intervals between the attacks. The duration of the disease is marked by years rather than months. In my own series of cases the average duration of symptoms is eight years. In reply to the query, " How long have you been ill ? " it is not uncommon for the patient to reply, " As long as I can remember."

The onset of the symptoms is insidious ; the patient notices that he has a feeling of discomfort two or three hours after the principal meals. Thus, a common history is, that

the discomfort is experienced about four or five o'clock in the afternoon, and continues until the next meal be taken. If dinner be taken in the evening, the discomfort may recur about ten o'clock. In the early stages of the ulcer the discomfort may be trifling, and little importance is attached to it, but it increases gradually, until it merits the description of pain. The frequency of its return likewise increases, so that after every meal there is the same sequence of events : pain coming on within two or three hours, continuing up to the time of the next meal, and relieved temporarily, by the taking of food. To the patient it appears as if the pain were due to want of food, not to the food taken some hours previously. For this symptom Sir Berkeley Moynihan has suggested the apt description " Hunger pain."

In the later stages of the disease a prominent symptom is pain coming on in the early hours of the morning. The patient goes to sleep in comfort, but is awakened about two or three o'clock, by a burning pain in the epigastrium. This early-morning pain is symptomatic of hyper-secretion.

A most marked feature of duodenal ulcer is the intermittence of the symptoms. They occur from time to time in attacks, and during the intervals between the attacks the patient may be in excellent health, and be able to enjoy an ordinary diet without sign of discomfort. The attacks may last a few days or continue for weeks. Often there is a definite cause for the onset of the attack—" a chill," some indiscretion in diet, or a change of climate.

One of my patients, who had had symptoms for nearly thirty years, told me that during his undergraduate days, while he was at Cambridge, he was always perfectly well, but directly he came to London for his vacation, he had an attack which continued as long as he remained in town.

Sometimes the intervals between the attacks are measured by years, so that the patient may think he is quite cured,

until he is disillusioned by a return of his trouble. Gradually the severity, or frequency of the attacks, or both, increases, and the intervals of good health become shorter. Even between the attacks the patient suffers from discomfort after food, accompanied by flatulence and belching of gas. Up to this point the patient may have maintained his normal weight and natural energy. But with increasing inability to take normal diet without pain, he restricts his food supply, begins to lose weight, and suffers in mental and bodily vigour.

SYMPTOMS

Pain.—The pain in duodenal ulcer usually is referred to the epigastrium. Radiation to other regions is uncommon. It is burning and gnawing in character, in the early stages, discomfort rather than pain, but in the later stages it may be very severe. Indeed, I have known patients in whom the pain was of such severity, that they were, for a short time, " doubled up " with it. This, however, is exceptional. My experience is that pain comes on rather sooner after a meal than is generally taught. I should put the usual time of onset at about two hours, sometimes it is delayed for five or even seven. In a few instances patients state that the pain comes on immediately after, or within half an hour to an hour of taking food. In some of these cases adhesions are present, or there is puckering of the duodenum, but this is not the case invariably. Possibly, in the early stages, the onset of the pain was not so rapid, but the later and more immediate pain has impressed itself so much on the patient's mind, that the memory of the earlier sequence of events has been obliterated. The pain of duodenal ulcer can be relieved, temporarily at least, by the administration of bicarbonate of soda.

Vomiting.—In 60 per cent. of my cases there is a history

L

of vomiting. Some writers state that vomiting is rare in duodenal ulcer, except when there is duodenal stenosis. With this statement I do not agree. I think that vomiting is not uncommon, but is rarely due to duodenal stenosis. It is due, either to reflex emptying of the stomach, or to reverse peristalsis of the duodenum, beginning at the site of the ulcer. Some years ago I suggested that the vomiting might be due to reverse peristalsis, and my hypothesis was characterised as being very improbable. Since then, on more than one occasion, by means of the X-rays, I have been able to see this reverse peristalsis commencing at the site of the ulcer, and, as it were, shooting the duodenal contents back into the stomach. Further evidence of such regurgitation is the observation, that in duodenal ulcer, it is not uncommon to find small quantities of bile in the fasting stomach.

As vomiting was present in 60 per cent. of my cases, and organic obstruction, as verified by operation, in only 20 per cent., it is clear, that in a considerable proportion, the vomiting is not due to organic stenosis.

Usually the amount vomited is not large in quantity; the emesis occurs two or three hours after food, and is followed by immediate relief from pain. When there is stenosis due to cicatrisation of the ulcer or duodenal adhesions, the patient may vomit large quantities, and there is marked food retention.

Hæmorrhage.—Hæmorrhage, as evidenced by hæmatemesis or melæna or both, occurred in 22 per cent. of my cases. It is usually a late symptom. Contrary to the accepted teaching, I believe that in the majority of cases hæmorrhage, when it occurs, comes, not from the ulcer, but from the mucous membrane of the stomach. The hæmorrhage is out of proportion to the extent and erosion of the duodenal ulcer. In such cases I have seen the whole of the gastric mucosa studded with numberless bleeding points. The

interior of the stomach is in the condition described as "weeping blood." I have several microscopic sections of the gastric mucosa in cases of duodenal ulcer, in which blood clearly can be seen escaping between the epithelial cells. Probably this condition of the mucosa is due to the irritation of hyper-acid gastric juice, or to the continual presence of gastric juice in the fasting stomach. In support of this view it is an interesting fact, that marked hyperchlorhydria was present in all but one of my patients who had had recent hæmorrhage.

As I have said before, as a rule, hæmorrhage is a late symptom, but occasionally an alarming hæmorrhage is the first symptom that attracts the attention of the patient. In these cases careful questioning will always elicit the fact, that there has been previous indigestion.

Examination of Gastric Contents.—Very often (in 50 per cent. of my cases) duodenal ulcer is accompanied by hyper-secretion, so that if a tube be passed in the early morning, from 20 c.c. to 50 c.c. of clear acid fluid is drawn from the stomach. That this is hyper-secretion, and not the result of obstruction, is proved by the circumstance that the fluid drawn off contains no food-remains, and that in these cases the stomach empties itself within the normal limit of time, and that the amount of the test-meal withdrawn is not greater than the average in health.

In my experience duodenal obstruction, due to adhesions or cicatrisation, is present in 20 per cent. of the cases. When this is so, food-remains are found in the stomach in the morning, together with a considerable amount of fluid, sometimes as much as 500 c.c. The amount, too, of gastric contents recovered after a test-meal is in excess of the normal.

I believe that at some period of the disease, hyper-chlorhydria is present in most cases of duodenal ulcer. In

some instances hyperchlorhydria is present during, or shortly after, the attacks, but between the attacks the free hydrochloric acid is normal, or a little above normal. Sometimes the hyperchlorhydria is latent, that is, there is excess of free hydrochloric acid, but its presence is masked by the regurgitation of bile from the duodenum into the stomach, which, as I have pointed out already, occurs, not infrequently, in cases of duodenal ulcer. In 52 per cent. of my cases there was hyperchlorhydria, and hyper-acidity in 70 per cent.

In a few cases (10 per cent. in my series) no free hydrochloric acid is found on examination of a test-meal.

I am of opinion that in these cases the absence of free hydrochloric acid is secondary to intestinal stasis, or to appendicular disease. I think we may state, that as a general rule, hyperchlorhydria accompanies duodenal ulcer, and that it is uncommon to find the free hydrochloric acid diminished.

Diagnosis.—In the vast majority of cases of duodenal ulcer, the diagnosis presents no difficulty. The history alone is sufficient. So far as diagnosis is concerned physical examination of the patient is of secondary importance.

Investigation of the gastric motility and secretion should never be omitted. It is true that in the majority of cases it is valuable only as a confirmation, but in a number of cases it is of the utmost value in the differential diagnosis.

As regards the history—as has been well emphasized by Dr. Christopher Graham—it is not the periodicity of the attacks nor the chronicity; both these characteristics are present in appendicular gastralgia; it is (i) the *time* of the onset of the pain and other symptoms, (ii) the *regularity* of the pain and other symptoms, (iii) the means by which the pain and other symptoms are relieved.

The important points in the diagnosis of duodenal ulcer are :

PLATE XXXIX.
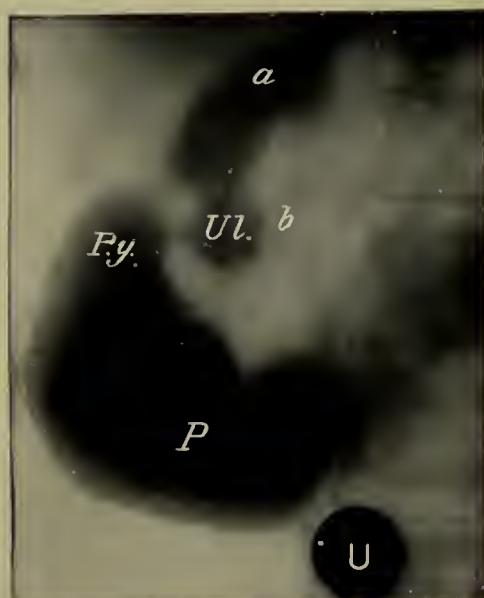
FIG. 55.—The pyloric portion of the stomach and the duodenum in a man aged 67, taken on the couch after bismuth, showing a chronic duodenal ulcer (*Ul.*) with raised margins. The duodenum is much dilated, especially in its first part above the ulcer. It showed active peristalsis, but the bismuth was returned repeatedly from the lowest to the highest part of the duodenum as each wave passed off. *P.*, pyloric portion of the stomach; *Py.*, pylorus; *Ul.*, base of the duodenal ulcer; *a*, *b*, first and second parts of the duodenum; *U*, umbilicus, marked by a penny. There was pyloric stenosis, and a series of reversed peristaltic waves was observed, passing from the pyloric end along the curvatures toward the fundus. After forty-eight hours there was still a large amount of bismuth in the stomach. At the operation a chronic duodenal ulcer was found just beyond the pylorus; the duodenum as a whole was much dilated, especially its first part, and there was much inflammatory thickening around the ulcer, extending up the duodenum. The ulcer was adherent to the pancreas.—By Dr. A. C. JORDAN.

1. The definite relation of the pain to food.
2. The length and constancy of the time interval.
3. The recurrence of the attacks.
4. The chronicity of the disease.
5. The intervals of good health between the attacks.
6. The relief of pain by alkalis.
7. The presence of hyperchlorhydria, hyper-acidity, and hyper-secretion.

The following is a good illustrative case of duodenal ulcer : A gentleman aged forty-three years began to suffer from attacks of discomfort in the abdomen twenty-seven years before I saw him. The attacks came on at intervals, and lasted two to three weeks at a time. During the attacks the discomfort came on regularly at five o'clock in the afternoon and lasted until dinner-time. He did not suffer much from flatulence. While he was out of London he was free from discomfort, but directly he came back, invariably he had an attack. Ten years before I saw him the attacks became more severe, so that instead of discomfort he had pain, which, during the attacks, came on from two to two and a half hours after food. For five years the attacks had recurred at longer intervals, but had been more severe, and the pain more continuous. Latterly, the pain had come on at any hour of the day or night; often he was awakened during the night by it, and was accustomed to take some bread and butter, after which he obtained relief for about an hour. Food relieved the pain immediately. He had never had any vomiting, but during the attacks had acid eructations and a desire to vomit. Between the attacks he was always absolutely well. Almost invariably the attacks were brought on by some definite cause, such as getting a chill, by worry, or by taking indifferent whisky. There was no tenderness anywhere in the abdomen.

The analysis of the gastric contents was as follows :

In the stomach in the morning after ten hours' fast—60 c.c.

Recovered one hour after test-meal—120 c.c.   Very good motility, some mucus, normal colour.

Total acidity	.	.	.	.	. 88
Total chlorides	.	.	.	.	. 0·430
Free HCl	.	.	.	.	. 0·058
Protein HCl	.	.	.	.	. 0·273
Mineral chlorides	.	.	.	.	0·098

Diagnosis : duodenal ulcer.

At the operation I found a puckered, cicatrised ulcer on the anterior wall of the duodenum close to the pylorus.   The appendix was chronically inflamed and was removed, and a posterior gastro-jejunostomy performed.   The patient was completely relieved, and now feels better than he has done for many years.

Occasionally hæmorrhage is the first symptom that attracts the patient's attention.

A man aged forty-three (admitted under the care of my colleague, Dr. Soltau Fenwick), noticed some years before admission that he was passing black motions.   Cross-examination elicited the fact that for many years previously he had suffered from indigestion.   Five years before admission he began to suffer from severe pain in the stomach three hours after food, sometimes accompanied by vomiting.   A few days before admission he began to suffer from vomiting one and a half hours after food, and again he noticed that his motions were black.   On admission he had some melæna, which continued with slight intermission for three weeks. He became extremely anæmic, but under medical treatment gradually improved.

The following is the result of the gastric analysis :

In the stomach in the morning after ten hours' fast—nil.

Recovered one hour after test-meal—165 c.c.

The contents were normal in colour, good motility, not a marked amount of mucus present. Volatile acids, 6·4.

Total acidity	.	.	.	.	. 100
Total chlorides	.	.	.	.	. 0·448
Free HCl	.	.	.	.	. 0·069
Protein HCl	.	.	.	.	. 0·281
Mineral chlorides	.	.	.	.	0·098

Differential Diagnosis.—The conditions which present the greatest difficulty in diagnosis are, gastric ulcer, gall-stones, appendicular gastralgia, and partial obstruction of the large bowel.

The differential diagnosis of gastric and duodenal ulcer has been discussed already (p. 151).

In **gall-stones** the pain has no relation to food, appears suddenly without warning, and disappears as suddenly as it comes. It is not relieved by food or alkalis. Vomiting gives no relief. The pain may be exceedingly severe (biliary colic), and cause spasm of the diaphragm. The pain is epigastric and radiates to the back and right scapula, or may surround the right half of the chest like a girdle.

Cholelithiasis in my experience is never accompanied by hyperchlorhydria; any change in the gastric contents is in the direction of diminished rather than of increased free hydrochloric acid, evidence of a secondary gastritis.

Vomiting is common in cholelithiasis, but unlike the vomiting in duodenal ulcer, is not followed by any abatement of the pain. In the early stage of gall-stone cases often there occur mild dyspeptic attacks, that is, irregular attacks of slight discomfort after food, accompanied by flatulence, sometimes coming on after food, sometimes not, appearing suddenly, disappearing as suddenly as they come. The irregularity of the onset of these attacks, the absence of any constant relation to the ingestion of food, render these

attacks very suggestive. In such cases gastric analysis will reveal little or no deviation from the normal.

In **appendicular gastralgia** the symptoms sometimes mimic duodenal ulcer with remarkable fidelity. There is often the same history of recurrent attacks, the same chronicity, and in rare instances, appendicular gastralgia is accompanied by hyperchlorhydria. Even " hunger pain " may be a symptom of appendicular disease, or to put it more accurately, I have met with cases in which " hunger pain " was a symptom, and in which there was a diseased appendix, but no discoverable duodenal ulcer.

It is possible of course, that in such cases an early duodenal ulcer may be present which is confined to the mucous coat, and so not palpable at operation. (See case on p. 157.)

The points which aid us in distinguishing the two diseases appear to me to be :

First, in duodenal ulcer, usually the patient is free from symptoms between the attacks ; in appendicular gastralgia, even between the attacks, the patient suffers from flatulence and discomfort after food.

Secondly, the radiation of the epigastric pain towards the lower part of the abdomen is highly suggestive, if not diagnostic, of appendicular trouble.

Thirdly, the discovery of tenderness over the appendix sometimes throws light on a doubtful case.

Fourthly, the pain of duodenal ulcer is more severe than that due to appendix disease. Sometimes, however, the sufferers from appendicular gastralgia have attacks which compel them to lie up. In some instances these attacks are accompanied by a rise of temperature which suggests at once the true cause of the symptoms. In other cases the temperature remains normal throughout the attack.

Fifthly, duodenal ulcer usually is associated with hyper-

chlorhydria ; in appendicular gastralgia the free hydrochloric acid is diminished or absent.

With regard to the appendix cases in which there is hyperchlorhydria, my impression is, that in duodenal ulcer hyperchlorhydria is associated with much more severe pain than is the case with hyperchlorhydria in appendicular disease.

On several occasions I have met with early cases of malignant growth of the **large intestine** in which the history suggested duodenal ulcer. The patient's chief symptom has been, pain coming on from two to three hours after food, and relieved by vomiting. A curious feature of some of the cases has been the periodicity of the attacks. In the cases which I have seen, cross-examination invariably has elicited the fact, that during the attacks the pain comes on quite independently of food and is not relieved by taking more food. Such cases illustrate the importance of a carefully taken and accurate history.

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CHAPTER X

THE TREATMENT OF GASTRIC AND DUODENAL ULCERS

The Treatment of Gastric Ulcer.—To Rydygier belongs the credit of having performed the first operation for the cure of a gastric ulcer. In the year 1881 he resected a gastric ulcer from the posterior wall of the stomach ; his patient recovered and was completely cured. In the year 1893 Doyen substituted gastro-jejunostomy for excision of the ulcer. This surgeon and Talma of Berlin, independently, had come to the conclusion that some form of pyloric stenosis, probably spasmodic, is the chief factor in maintaining the hyperacidity which prevents the healing of gastric ulcers. Gastrojejunostomy was soon adopted as the operation of choice for gastric ulcer. Rydygier, however, continued to advocate excision, in the belief that the scar of an ulcer may become the seat of carcinoma. The results of gastro-jejunostomy, when performed for pyloric stenosis, were brilliantly successful. To some extent, gastro-jejunostomy for ulcers without stenosis proved disappointing. It was thought that the failures were due to the patency of the pylorus. Some surgeons, therefore, advocated that when gastro-jejunostomy was performed in such cases, the pylorus should be excluded deliberately by suturing, a measure based on erroneous pathology and little calculated to prevent closure of the anastomotic opening. Doubtless some patients were unrelieved on account of mistakes in technique, but probably many more were no better because gastro-jejunostomy was performed without proper indication. The operation was

performed on a clinical diagnosis, and not on the evidence found on the operation-table. Before operation a diagnosis of gastric ulcer was made, at the operation no ulcer was found. It was assumed that the symptoms were due to an ulcer of the mucous coat, without induration, and so not palpable. Gastro-jejunostomy was performed, and a lesion in the appendix or elsewhere was undiscovered and left untreated. The patient was no better, the case being recorded as an operative failure, whereas it was really, as Dr. W. J. Mayo puts it, " a mistake both in diagnosis and operation."

During the last few years the treatment by excision has been revived, but it is advocated for different reasons than formerly. It is argued, first, that gastro-jejunostomy is a drainage operation, and therefore its performance in cases of ulcer away from the pylorus is useless, as the food continues to pass through the patent pylorus and not through the new opening. In other words, the drain does not act. I have already expressed my firm conviction that gastro-jejunostomy is a physiological, and not a drainage operation. Whether the food passes out through the pylorus, or through the anastomosis, is of no consequence. I have performed gastro-jejunostomy many times in cases of ulcer of the lesser curvature, or ulcer at the cardiac end of the stomach, and have kept in touch with all the patients who had ulcers in these positions, and I can say, without hesitation, that the results have been more satisfactory than the results of gastro-jejunostomy for other ulcers. Every one of these patients has been completely relieved.

Secondly, with regard to callous ulcers at the pyloric end of the stomach, it is said that these should be excised, because they are apt to degenerate into cancer. It is stated that a large proportion of gastric carcinomata have their origin in simple ulcer. The treatment by resection, in selected cases, has received the imprimatur of so great an

authority as Dr. W. J. Mayo. The experience at St. Mary's Hospital (Rochester) is that in no less than 71 per cent. of the cases of gastric cancer, the starting-point is a gastric ulcer. From personal knowledge I have such a high admiration and respect for all the work of the Mayo clinic, that I feel it is almost presumptuous to disagree with any conclusions arrived at by Dr. Mayo.

I do venture to think, however, that the evidence, on which the existence of ulcer previous to malignant disease is based, is far from conclusive. If the view that cancer be grafted on ulcer be correct, gastro-jejunostomy must be performed often for ulcers which already are malignant. Why is it, then, so rare an event for a patient to die from cancer, after gastro-jejunostomy for supposed simple ulcer? When a mistake is made, it is more likely to be a mistake in the opposite direction. It is the supposed malignant case which proves innocent, not the supposed simple case which proves malignant.

Most surgeons who have had much experience in gastric surgery have met with cases in which there is a hard mass at the pylorus, tightly bound down to the pancreas and other structures, with enlarged glands along the lesser curvature, a condition of things rendering resection absolutely impracticable. A gastro-jejunostomy is performed as a palliative operation, in the hope that the patient will gain a few months' comfort. To the surprise of the surgeon the patient has regained perfect health, and lived so long that the existence of cancer is negatived. Yet if the grafting hypothesis be correct, it is in just such a case as this, that one would expect death eventually to occur from cancer.

I have had a fairly large experience of the after-results of gastro-jejunostomy, both as performed by myself and as performed by others, and I have met with two cases only (one operation performed by myself, and one by another

PLATE XL.
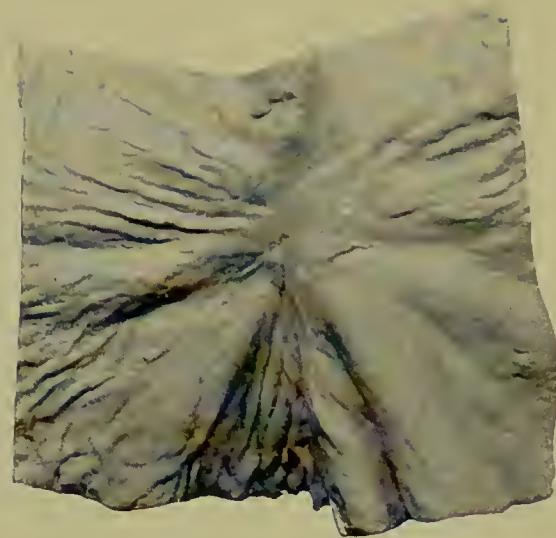
FIG. 56.—Cicatrix of a chronic ulcer.
(Soltau Fenwick.)

FIG. 57.—Recurrent ulcer in the operation scar, from
a man who died of hæmatemesis three months
after excision of gastric ulcer. (St. Bartholomew's
Hospital Museum, No. 1917b.)

surgeon) in which, after gastro-jejunostomy for supposed simple ulcer, the patient has died from gastric carcinoma. Professor Kocher has published lately a review of fifty cases of gastro-jejunostomy, in which the periods which have elapsed since operation, varied from two to twelve years. There was no recurrence in any case, and Dr. Kocher concludes with justification that there is therefore no need for resection. Dr. Gressot states that malignant degeneration of surgical ulcers occurs in only 2·3 per cent. of all cases after gastro-jejunostomy. In view of these observations, it appears to me difficult to accept the hypothesis that carcinoma is grafted on simple ulcer in such a high percentage of cases as is taught at the present time. (See also Chap. XVIII).

Personally, I believe that gastro-jejunostomy is preferable to excision, the risk of which is undoubtedly greater than that of the short-circuiting operation.

Further, judging from recorded cases and from my own experience, there is little doubt that relapses are much commoner after excision than after gastro-jejunostomy.

This is just what one would expect, because excision removes the ulcer, but does not influence the causal factor of ulceration. This, in my judgment, is the chief objection to excision.

Gastro-jejunostomy, therefore, should be the operation of choice, because it is a physiological operation, and so alters the gastric contents, as to promote healing of the ulcer.

I will now attempt to answer the question, "When should surgery interfere in cases of gastric ulcer?" (Acute ulcer, apart from the complications of gastrorrhagia and perforation, does not call for surgical measures.) The answer to this question appears to me to depend on three considerations :

1. **What is the Natural History of Gastric Ulcer treated Medically?**

2. The Mortality of the Surgical Treatment Proposed.

3. The Prospect of Cure after Operation.

1. The Natural History of Gastric Ulcer.—The mortality from gastric ulcer has been estimated variously by different writers, some placing it as high as 50 per cent., and others as low as 20 per cent.

The important point is to determine what proportion of the cases considered to be " cured " remain perfectly well. In my Hunterian lectures in 1906 I gave an analysis of the after-results of a series of cases of gastric ulcer ; put in tabular form the results were as follows :

```
" Not cured : "
    Signs of gastric ulcer  .     .     .     .     .     .     .  40
    Operation subsequently necessary  .     .     .     .   5
    Died from gastric disease  .     .     .     .     .     .   1
                                                          —  46
    " Cured "  .     .     .     .     .     .     .     .     .   . 19
    " Probably cured " .     .     .     .     .     .     .   2
    " Probably not cured "   .     .     .     .     .     .   5
                                         Total     .  72
```

To avoid any possible exaggeration I will include the five " probably not cured " patients among the " cured." Even so, 64 per cent. of the patients who have been traced have been uncured by medical treatment. Looking at the figures in the most favourable light, at least 31 per cent. of the patients discharged as cured have relapsed, while if the condition of the untraced patients be as unsatisfactory as is that of those who have been traced—and there is no reason to suppose that it would be better—the proportion of re-lapses would be more than double this figure. When I point out that these patients were in hospital on a strict milk diet for an average period of six weeks, and that they were warned to keep on a milk diet for a further period of six weeks, and to exercise great care for three more months, the significance of such a large proportion of relapses is

apparent. But the worst has not been said. Although nineteen of the patients are classified as cured, the use of the term is relative, not absolute, and only implies that for the present these patients are free from symptoms of gastric ulcer. One of them, although quite well at the time of inquiry, had been in hospital again for six months, while another was an in-patient for a shorter period. Only seven of the nineteen patients have remained free from any symptoms of gastric ulcer since they left hospital. Of the seventy-five patients whom we have been unable to trace, it is noted that two had been in hospital twice before, and ten once, so that of the untraced patients eleven—*i.e.* 14 per cent.—were the subjects of relapsing gastric ulcer.

Considering that this series does not include those patients admitted with perforated gastric ulcer, or those admitted with pyloric stenosis, or other sequels of gastric ulcer, I think we may conclude, that so far as hospital patients are concerned, between one-half and two-thirds of the serious cases relapse, and that the proportion of real cures is under 25 per cent.

Gastric ulcer in private practice presents a totally different picture. Private patients, amid more favourable surroundings, bear more readily the necessary restriction of their diet, and relapse is much less common. Consequently operation is required less often for the treatment of an existing ulcer, as for those cases in which, owing to neglect to commence treatment early, cicatrisation of the ulcer has produced pyloric stenosis, gastric adhesions, or hour-glass stomach.

2. **The Mortality of the Surgical Proceeding Proposed.**—My view is that the best method of treating an intractable gastric ulcer is by the operation of gastro-jejunostomy.

The mortality of this operation in skilled hands is not greater than 3 per cent. When it is realised more fully

that gastro-jejunostomy does not interfere with the metabolic processes of the human body, and that after it patients may live for years in perfect health, doubtless these relapsing cases will be dealt with surgically at an earlier stage, and we may hope reasonably that the mortality will be further diminished. Indeed, there is no reason why the operation of gastro-jejunostomy, if performed before the patient has been worn out and emaciated by months or years of suffering, should be attended with a greater mortality than the operation of appendicectomy during a quiescent interval.

3. **The Prospect of Cure by Operation.**—Some years ago I collected 278 cases of gastro-jejunostomy from surgical literature and endeavoured to ascertain the after-results. The after-history was obtained in 143 of the cases, and the results are shown in the following table :

" Cured " of gastric trouble :
    Alive and well . . . . . . $\left.\begin{array}{c}115\\3\end{array}\right\}=118$ (82 per cent.).
    Died of other trouble . . . .
Unsatisfactory cases :
    Died subsequently . . . . . $\left.\begin{array}{c}4\\5\end{array}\right\}=9$ (6 per cent.).
    Relapsed . . . .
Fairly good results :
    Occasional dyspepsia . . . . . $\left.\begin{array}{c}9\\7\end{array}\right\}=16$ (11 per cent.).
    Some care in dieting necessary . . .
$$\overline{143}$$

When it is remembered that these operations were performed by many different operators, and for various conditions, the results must be considered highly satisfactory.

Investigation of my own cases gives a very similar result : 81 per cent. of my patients have been relieved completely ; 12 per cent. have been greatly improved, that is, have been completely relieved of their pain, but suffer from occasional discomfort, or have to be careful as to their diet ; and 5 per cent. either have been unrelieved, or have had recurrence of their symptoms.

When should Surgery interfere in Cases of Chronic Gastric Ulcer?—We have now seen that the mortality from gastric ulcer treated medically is at least 20 per cent., and further, that at least 50 per cent. of the so-called " cures " relapse, while probably not 25 per cent. of the patients treated medically are really cured. On the other hand, gastro-jejunostomy holds out a great prospect of cure, the proportion of relapses being probably not more than 10 per cent. The immediate risk of operation is about 3 per cent., to which may be added the remote risk from jejunal ulcer of under 2 per cent. I realise fully the danger of advocating operation from comparisons of this sort, but at least these figures suggest that the question of surgical treatment should be considered seriously in every case which fails to yield to medical treatment.

Leube says that if a gastric ulcer be not cured by four or five weeks of treatment, it will not be cured by medical treatment alone. Allowing a wider latitude than this, I would formulate the principles on which to act, as follows : If, after six weeks' complete rest, on a milk diet, a further period of six weeks on a milk diet with comparative rest, followed by three months' careful dieting, the patient be not free from definite symptoms, or if, after apparent cure, the patient have a relapse, operation is probably in the best interests of the patient. The symptoms, however, must be clear and definite. The performance of gastro-jejunostomy in cases of vague gastric disorders, unaccompanied by definite clinical or chemical signs, cannot but lead to the discrediting of a valuable, and in suitable cases, legitimate operation.

The Treatment of Duodenal Ulcer.—In duodenal ulcer the indication for surgical treatment is much more definite than in gastric ulcer. We have evidence that a gastric ulcer may cicatrise and give rise to no further symptoms. In duodenal

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ulcer, such a favourable issue is rare, if it occur at all. My experience is that when once a patient has marked symptoms of duodenal ulcer, recurrent attacks of pain coming on two hours or more after food, with hyperchlorhydria, he will be subject to periodic return of symptoms until surgical treatment be adopted. Dieting, medicinal treatment, and gastric lavage will give relief but will not bring about a cure. There is no doubt that a duodenal ulcer may heal, but this does not necessarily mean relief. The process of cicatrisation often causes stenosis. Even if the ulcer be small, and no narrowing of the duodenum follow the process of healing, the symptoms appear to continue. I have operated in a number of cases in which I found a small healed ulcer, but yet hyperchlorhydria was present and the patient had continued to suffer from typical periodic attacks of duodenal ulcer pain. Recently, I operated on a young woman who had been in hospital for six weeks, and had been discharged, apparently quite well. She returned three months later with recurrence of her former symptoms. At the operation there was a small scar in the duodenum, not larger than a threepenny piece. There was no stenosis, and no other lesion discoverable. Why in such cases the symptoms persist, is difficult to understand, unless it be that the presence of a scar sets up pyloric spasm.

Another factor which renders operation in duodenal ulcer more imperative than in gastric ulcer, is, the greater risk of perforation. On account of the thinness of the duodenal wall, perforation is a more likely complication than is the case in gastric ulcer.

In the early stages medical treatment should have a thorough and extensive trial. Milk diet, rest in bed, the administration of half an ounce of olive-oil three times daily before meals, as recommended by Professor Matthieu, should be persevered with for at least six months. At first, pro-

bably the ulcer is limited to the mucous coat, and if treated during this stage, we are justified in acting on the belief that it may heal and cause no further trouble, although we do not know whether such is the case or not. (See case on p. 157.)

If, in spite of treatment, the symptoms continue, or if, after apparent cure, relapse ensue, only surgical treatment will give relief.

The following operations may be performed :

1. Gastro-jejunostomy.

2. Excision of the ulcer, if it be small and free from adhesions.

3. Finney's operation.

4. Resection of the portion of duodenum in which the ulcer is situated.

5. Resection of a portion of the duodenum with the ulcer, closure of the distal end of the duodenum, and implantation of the proximal end of the duodenum into the side of the distal portion.

Sir Berkeley Moynihan has advocated excision in suitable cases. This method is open to the same objection as resection for gastric ulcer. It removes the ulcer, but does not influence the hyperchlorhydria.

The results of gastro-jejunostomy for duodenal ulcer are so satisfactory, that I think it is preferable to any method at present in use.

The argument for resection of gastric ulcers, that the ulcer may become the site of cancer, does not apply to ulcer of the duodenum, as cancer of the duodenum is exceedingly rare.

Dr. W. J. Mayo and Sir Berkeley Moynihan recommend that when gastro-jejunostomy is performed, the ulcer should be infolded by two or three mattress sutures, to promote healing and guard against hæmorrhage. This addition can

do no harm, but whether it is necessary is an open question. I do not adopt it as a routine procedure, although I have used it in some cases. I have never yet had a case of hæmorrhage from the ulcer after gastro-jejunostomy.

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CHAPTER XI

THE COMPLICATIONS OF GASTRIC AND DUODENAL ULCERS

1. PERFORATION

A. Perforation of Gastric Ulcer

In the field of human disease there is nothing more tragic than death from sudden perforation of a gastric ulcer—tragic not only from its suddenness, but from the circumstance that the victim is often young, in the prime of life, and to all outward appearance in the enjoyment of perfect health. The measure of success which has attended the efforts to snatch these victims from the very jaws of death, must be accounted one of the greatest triumphs of surgery.

Perforation of the stomach may be acute or chronic.

In **acute perforation** an ulcer gives way suddenly, allowing the escape of gastric contents. In about 96 per cent. of the cases of acute perforation, the gastric contents escape freely into the general peritoneal cavity, and general peritonitis ensues. In about 4 per cent., owing either to the small size of the perforation, or to the rapid closing of the opening by a plug of lymph or omentum, the escape of gastric contents is limited. Such cases are described as sub-acute. Although the initial symptoms may be as severe as those in which there is general extravasation of gastric contents, the prognosis is more favourable, and the resulting peritonitis may be of very limited extent.

In **chronic perforation** the destruction of tissue takes place more slowly, so that by the time the whole thickness of

the gastric wall has been destroyed, the general peritoneal cavity is shut off by a barrier of plastic exudation. Thus, the escape of gastric contents is circumscribed, and the suppuration that ensues almost inevitably, is confined within a limited cavity, forming a peri-gastric abscess.

Although acute perforation does occur in cases of acute gastric ulcer, it is more common in the chronic form, owing either to absence of plastic peritonitis, or to sudden yielding of adhesions which have been formed.

Acute Perforation.—The frequency of perforation has been estimated by various writers at from 5 per cent. to 28 per cent. of all cases of gastric ulcer. In a series of 500 cases I find that perforation occurred in 12 per cent. Perforation proved fatal in 7 per cent. of the cases, a figure corresponding exactly to that given by Welch. Perforation is more common in women than in men (79·3 per cent. to 20·7 per cent.). Allowing, however, for the greater frequency of gastric ulcer in women, perforation is relatively more frequent in men. Thus, perforation occurred in 23 per cent. of the male patients, but in only 9 per cent. of the female.

In considering these figures we must bear in mind that until comparatively recently a clear distinction has not been drawn between gastric and duodenal ulcers, and so probably, a number of the ulcers classified as gastric really were duodenal.

Perforation occurs much more frequently in an ulcer of the anterior than of the posterior gastric wall. The importance of this is, that in 90 per cent. of the cases, the perforation is in a position readily accessible for surgical treatment.

SITE OF PERFORATION.
112 Consecutive Cases of Perforated Gastric Ulcer.
Anterior Wall.

Pyloric Third.	Middle Third.	Cardiac Third.	Posterior Wall.	Not stated.
32	33	32	12	3

PLATE XLI.
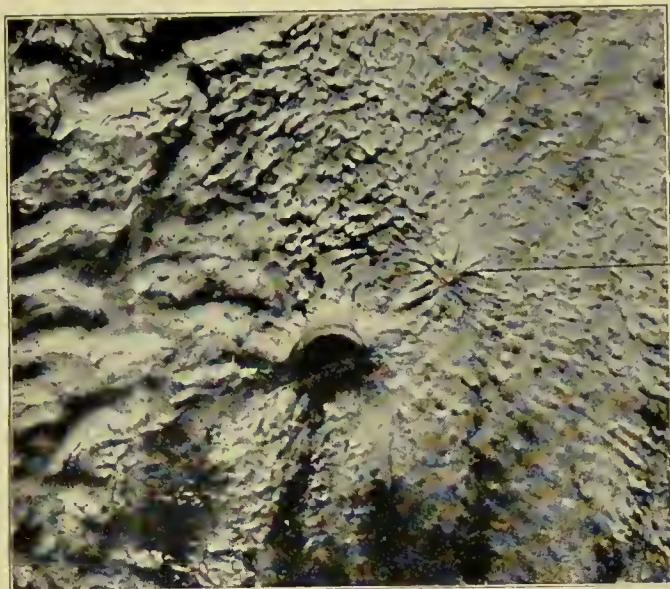
FIG. 58.—Acute perforating ulcer of the stomach in a
girl. To the right of the ulcer is a small cicatrix
which was discovered by photography. (Soltau
Fenwick.)
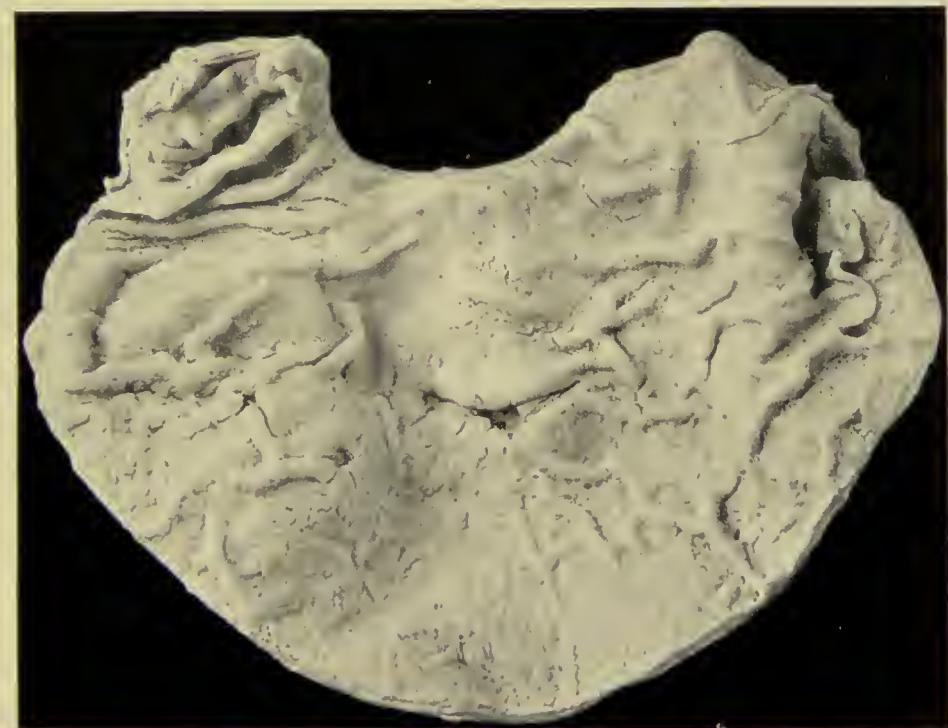
FIG. 59.—Perforation of a gastric ulcer in an infant. (Porter Parkinson.)

It is noteworthy that of the perforations in the anterior wall only three were near the greater curvature ; all the others were situated in the upper half of the anterior wall, and mostly near the lesser curvature. The site of perforation differs in the two sexes ; in the male the area of perforation is near the pyloric end of the stomach, while in the female perforation occurs more often towards the cardiac end of this viscus.

A second ulcer was present in 23 per cent. of this series of cases. The size of the perforation varies from a mere pin-prick to the size of half a crown, the average being about the size of a threepenny piece.

The suddenness of the onset, and the severity of the symptoms, depend probably in a very large measure on the size of the aperture, and the quality and quantity of the gastric contents. In one of the most acute cases I have seen, perforation occurred two hours after a meal of sausages and potatoes. Although operation was performed within six hours, the whole abdomen was filled with a purulent fluid which gave cultures of streptococci.

The Previous History.—It is a common belief that a considerable number of the patients who are the victims of perforation, have not suffered previously from indigestion. According to some writers the proportion of such cases is as high as 64 per cent. This is not in accord with my experience. In the great majority of cases, a history of previous gastric trouble can be obtained, if careful inquiry be made. I have met with only one instance in which, on cross-examination, no indications of previous gastric trouble could be elicited.

In the majority of cases the onset of perforation is sudden and unexpected, but in some (about 17 per cent.) there are premonitory symptoms, in the form of increased epigastric pain, with or without vomiting.

The Signs and Symptoms of Perforation.—In the litera-
ture of the subject there is perhaps no more vivid picture of
perforation of an ulcer than the historical account of the
case of Henrietta, Duchess of Orleans, which has been handed
down to us by one of her maids-in-waiting. This interesting
story is as follows :

"She had ailed for some time from pain in the stomach and
side, but seemed to be otherwise in good health. One day after
dinner she complained several times of pain. She drank some
succory water as a stomachic, and, putting the cup down with
one hand, with the other she pressed her side, and said in a
voice which betokened much suffering : 'Ha ! what a stitch in
the side ; what pain ! I cannot bear it.' Speaking these words,
she flushed, and a moment afterwards turned pale with a wan
lividity which astonished everybody. She kept crying out, and
begged to be carried away, for she could not longer hold up.
Supported by the arms of others, she managed to walk, but with
difficulty, and bent double. Put to bed, she cried out more than
ever that the pain in the pit of her stomach was past belief.
She turned from side to side. A physician was fetched, who
pronounced her complaint to be colic, and who prescribed
suitable remedies. But the pain continued. She said that her
suffering was greater than could be conceived, and that she
would die.

"All this occurred in less than half an hour. Whatever she
swallowed made her retch ; she brought up only a little mucus
mixed with food. The efforts to vomit and the excessive pain
threw her into a state of exhaustion which resembled repose, but
she told the bystanders not to deceive themselves ; that the pain
was as great as ever, and that she had no strength left to cry
out. She heard someone remark that she was easier, and she
said : 'That is so far from being true that were I not a Christian
I would kill myself, so great are my sufferings. It is wrong to
wish evil to anyone,' she added, 'yet I would that somebody
could feel for a moment what I feel, so as to know what my

pain is like.' Her pulse became imperceptible, her limbs cold. Her friends anxiously asked if nothing more could be done; they suggested a score of remedies, and at last her physicians in sheer desperation made an attempt to bleed her, but the blood would not flow. They gave her some broth, for she had taken no food since dinner. She no sooner swallowed it than her sufferings increased. She complained that her stomach was filling up. Death was painted on her face. The last struggle was short, and after two or three convulsive movements about her mouth, she died nine hours from the beginning of her illness."

Suddenness of onset is the most characteristic feature of acute perforation. Suddenly the patient is seized with intense abdominal pain. In some instances, at the time of the seizure, the patient experiences a feeling as of something giving way. At first the pain is limited to the epigastrium, but soon it becomes general, and is referred to the whole of the abdomen. It is so acute and agonising as to be almost unbearable, and the patient's face bears witness to the severity of his sufferings. The extremities are cold, beads of perspiration appear on the forehead, the respirations are quick and shallow, and the patient replies to questions in short, jerky sentences. The body temperature is subnormal, often as low as 95° F. or 96° F. At first the pulse-rate is quickened, and its volume poor, but if the patient be not seen until the first shock of the seizure has passed off, the pulse-rate may be quickened little, if at all, and the volume good. The abdomen is retracted, and there is a " board-like rigidity "—a sign most significant of perforation of a hollow viscus. This rigidity is most marked over the upper part of the recti muscles, and sometimes is limited to this region. Usually there is tenderness of the upper part of the abdomen; the gentlest pressure induces protective spasm of the abdominal muscles at once.

As a rule, the point of maximum tenderness corresponds to the site of perforation. Absence of liver dullness, as determined by percussion, is a sign of no practical importance. Authorities differ as to the frequency of vomiting. In my experience it is present in about half the cases. Such are the early symptoms of perforation. In rare instances the inaugural shock is so profound as to prove fatal immediately (Maurice, Moynihan).

Often the symptoms of collapse pass off, the pulse improves, increasing in volume and decreasing in frequency, and the temperature may regain the normal. The pain continues, less agonising maybe, but no less constant. The improvement, however, is short-lived. Gradually but surely, fresh symptoms appear, harbingers of peritonitis caused by the presence of gastric contents in the peritoneal cavity. First and most important, there is a steady and progressive increase in the pulse-rate, gradually too its volume diminishes until it becomes the small, rapid, running pulse indicative of advanced septic peritonitis. The temperature may rise to 100° F. or 101° F. ; the retraction of the abdomen is succeeded by gradually increasing distension ; the abdomen becomes tense and tympanitic ; the respiration more and more thoracic, the extremities cold, the face pinched and cyanosed, and the patient slowly sinks, life rarely being prolonged more than a few days.

The following is an illustrative case :

A woman, aged thirty-five years, had suffered from epigastric pain for two years. At first the pain used to come on from half to one hour after food, but latterly it had had no definite relation to food, but had been worse at night. Sometimes the pain was relieved by food, but it was relieved always by vomiting. The vomit was very ' acid ' and frothy, and dark in colour. One morning at 8 A.M. she was seized suddenly with severe abdominal pain and vomiting. Three and a half hours later she was ad-

mitted into the hospital.   On admission she was extremely
collapsed, cold and blanched, and apparently in great pain.
Her temperature was 97° F., pulse-rate 140, small and feeble.
Immediately after admission she vomited two pints of material
containing some blood.   Her abdomen was distended, with
marked rigidity and tenderness in the upper part, especially to
the left of the middle line.   While preparations for operation
were in progress, a pint of saline was run into the rectum.   Open
ether was administered, and I opened the abdomen.   There was
much turbid fluid free in the peritoneal cavity.   The stomach
was very large, and was divided into two compartments by an
hour-glass constriction at the junction of the pyloric with the
middle third.   Immediately to the distal side of the constriction,
close to the lesser curvature, there was a perforated ulcer, the
size of a sixpenny piece.   The perforation was sewn up with two
rows of sutures.   Anterior gastro-jejunostomy was performed on
the proximal side of the constriction, a large drainage tube was
placed in Douglas' pouch and brought out through a stab incision
above the pubes, and the wound was sewn up.   The operation
lasted twenty-two minutes.   During the operation, and again
while being carried back to bed, the patient stopped breathing,
but recovered upon artificial respiration being performed.   After
operation the patient was in a condition of extreme collapse,
very cold, pulse 140, almost imperceptible.   Saline was given
by the axillæ and by the rectum, and thanks to the devoted
nursing of the Sister-in-charge she rallied gradually and made
an uneventful recovery.   A year later she had gained over three
stone in weight, and wrote, " I don't remember ever feeling so
well as I do at the present time.   I can eat almost anything."

The Diagnosis of Perforation.—When signs of general
peritonitis are present, the diagnosis presents no difficulty.
It should be our aim, however, to make a diagnosis and to
act upon it at a much earlier stage, but this is not always
easy, as is shown by the mistakes made by surgeons of
great experience.

In a few cases primary collapse is succeeded by a period of reaction, so that when the patient is seen his general condition may be such as to lead to doubt as to the existence of so serious a lesion as perforation of an ulcer. The pulse-rate may be not more than 80, its volume good, the temperature not raised, and beyond a little tenderness, nothing is discovered on examination of the abdomen. It is of the utmost importance to realise and to remember, that a normal pulse and temperature do not negative the existence of perforation. Decision in such cases is beset with difficulty. There is little but the history to guide us, and if we rely on this, we may perform an unnecessary operation; on the other hand, if we delay, the lull may be succeeded by a storm which tells us, unmistakably, that intervention is too late.

Difficulty in diagnosis is likely to arise especially in cases in which morphia has been administered. In dealing with cases of supposed perforation, it is a golden rule, never to give morphia except under one of two conditions: first, when immediate operation is decided on; secondly, when a diagnosis of perforation has been excluded absolutely. As soon as it is decided to operate, the morphia and atropine, always injected before the administration of ether, should be given at once. The patient is relieved of pain, and probably his general condition will have improved considerably by the time the preparations for the operation have been completed.

Although from time to time difficult cases are met with, in the vast majority it is possible, with care, to make a diagnosis of perforation. The important points are:

(i) A history of previous indigestion.

(ii) The sudden onset of agonising pain.

(iii) Rigidity of the upper part of the recti muscles. In the rare instances in which rigidity is absent, careful in-

spection always will reveal impaired mobility of the upper part of the abdomen.

Differential Diagnosis.—The chief diseases which give rise to difficulty in the diagnosis are: early pneumonia or diaphragmatic pleurisy, acute ptomaine poisoning, acute appendicitis, biliary colic, ruptured tubal gestation, and acute pancreatitis.

Of these, the first two are by far the most important, not only because sometimes the differential diagnosis is very difficult, but because in these diseases an operation is absolutely unnecessary and harmful.

In acute appendicitis, ruptured tubal gestation, and in acute biliary colic, surgical treatment is indicated, and so an exact diagnosis is of less importance.

In Early Pneumonia or Diaphragmatic Pleurisy, the most important diagnostic point is the marked increase in the rate of respiration in relation to the pulse-rate. A respiratory rate of thirty-four or more to the minute with a pulse-rate of a hundred or less, suggests a thoracic and not an abdominal lesion. In pneumonia or pleurisy there is impaired movement of the lower part of the chest, and the abdominal tenderness and rigidity is far less marked than in perforation. The rigidity is not continuous, the muscles may be induced to relax by gradual, firm pressure with the hand, or by instructing the patient to take a slow, deep expiration.

Acute Ptomaine Poisoning may simulate perforation so closely, that at times it is well-nigh impossible to distinguish between the conditions. The points that may be of service are: the possible history of others being attacked in the same way, the occurrence of diarrhœa, and the character of the vomit, which, in ptomaine poisoning, consists usually of undigested food, without that copious secretion of gastric juice and bile which characterises the vomiting, when present, in perforation.

A woman, aged twenty-six years, was seized, while out walking, with severe abdominal pain, and fell down in a state of collapse; her extremities were cold, temperature 97·4° F., pulse 80, respirations 28. She was well enough to give a clear account of herself, and it was ascertained that she had been in hospital three times for attacks of abdominal pain.

There was some rigidity of both recti above the umbilicus, and apparently pain on palpating the upper part of the abdomen. She vomited slightly after admission. A few hours later her pulse became poor and irregular, and it was thought that she was suffering from a perforated gastric ulcer.

I opened the abdomen, but found nothing to account for her severe symptoms. After she had recovered, we discovered that she was much addicted to drink, and apparently her symptoms were the result of a drinking bout.

In Acute Appendicitis, the rise of temperature, coincident with the initial onset of pain, usually will distinguish this affection from perforation of an ulcer, in which there is at first a fall of temperature. It is important to ascertain whether the pain *began* in the epigastrium, or in the appendix region.

Some years ago I was asked to see a young man who was seized suddenly with severe abdominal pain. His temperature was subnormal, pulse 96, and there was extreme tenderness and rigidity over the epigastrium. A diagnosis of perforated gastric ulcer was made, but a small incision above the umbilicus showed that there was no gas or extravasated fluid in the peritoneal cavity. Some months later the patient had a somewhat similar attack, but on this occasion there was extreme tenderness over the appendix. After the acute attack had subsided the abdomen was reopened and a diseased appendix removed. He had evidently had previous attacks of appendicitis.

I have little doubt that when I operated on him, his

symptoms were really due to acute appendicitis.   The mimicry of gastric perforation was remarkable, and I think in such a case a correct diagnosis is impossible.

It is well known that chronic appendicitis may mimic gastric ulcer closely, but this is the only instance [1] with which I am acquainted in which an acute attack of appendicitis has simulated perforation of a gastric ulcer.

In Biliary Colic the pain may be of agonising intensity, but it is rarely accompanied by such severe initial collapse. Usually the temperature is normal, but there may be pyrexia. Hiccough is commonly a distressing symptom, and vomiting is frequent.   The attack is often ushered in by shivering or by a rigor.   The pain is referred to the right hypochondrium, and often there is radiation to the right scapular region.   In cholelithiasis there may be a history of discomfort and flatulence after food, but not the definite and regular relation of pain to the ingestion of food which obtains in ulcer.   In biliary colic there is a history of previous attacks, some of which may have been accompanied by jaundice.

In Ruptured Tubal Gestation the history of previous amenorrhœa will give a clue to the correct interpretation of the symptoms, but it must be borne in mind that a history of vomiting of pregnancy may suggest gastric ulcer.   In ruptured tubal gestation, rigidity is more marked in the lower part of the abdomen, and it rarely is so pronounced as in perforation of an ulcer.   In tubal gestation the maximum tenderness is below, in perforation above, the umbilicus.   As in both these conditions surgical treatment is required, exact differentiation is not essential previous to operation.   On opening the abdomen, the presence of blood in the peritoneal cavity points at once to the presence of a ruptured tubal gestation.

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[1] I have operated since in a case of acute appendicitis, in which a severe attack of hæmatemesis suggested at first a diagnosis of gastric ulcer.

In Acute Pancreatitis the pain is severe, and is referred to the epigastrium, but as a rule there have been no previous symptoms suggesting an ulcer. From the first the pulse-rate is very rapid, vomiting is frequent, and the general condition of the patient very alarming considering the short duration of the illness.

In cases of doubt I hold very strongly that it is better to open the abdomen than to delay, provided that the surgeon definitely make up his mind to limit his operation to the making of a simple incision in order to determine whether there be free fluid, or gas in the peritoneal cavity. If the surgeon be content with this, he will do no harm. The danger is that he may proceed to search for some other seat of mischief, and instead of being a simple procedure, un-attended by serious risk, the operation may be so prolonged as to jeopardise the patient's life. In cases of doubt there must be no indecision. The surgeon must resolve firmly, to make only a small incision, and to close the abdomen immediately, if there be no free gas and no extravasated fluid in the peritoneal cavity. On no account should the operator allow himself to begin an extensive search for some other condition, unless the indications be very definite.

B. Perforation of Duodenal Ulcer

Perforation of an Acute Duodenal Ulcer is an occasional occurrence. Acute duodenal ulcers usually are of infective or toxic origin, and are associated with extensive cutaneous burns, or with acute appendicitis. I have had one case in which an acute duodenal ulcer perforated a few days after operation for acute appendicitis.

As a rule, patients with acute duodenal ulcer are so ill, that any operation is out of the question, even if the existence of a perforation be recognised.

Perforation of a Chronic Duodenal Ulcer is found usually on the anterior wall of the duodenum within an inch and a half of the pylorus. I have met with one instance of perforation of an ulcer on the posterior wall of the second portion of the duodenum, a position very difficult of access.

Perforation of a duodenal ulcer is a more serious catastrophe even than perforation of a gastric ulcer. In a few cases of perforated gastric ulcer, the aperture becomes closed by a plug of lymph or omentum. Rare as is such a happy issue in perforated gastric ulcer, it is more rare after perforation of a duodenal ulcer.

The clinical picture of perforated duodenal ulcer is very similar to that of perforated gastric ulcer. There is the same sudden onset of agonising pain, a similar board-like rigidity of the abdominal walls, a similar progressive sequence of untoward symptoms, the same urgent indication for surgical treatment, and a like fatal issue if no operation be performed. Little, therefore, need be added to what has been written already in the previous part of this chapter.

Perforation of a duodenal ulcer has to be distinguished from the same acute conditions as in the case of perforated gastric ulcer. As a rule, the differential diagnosis between a perforated gastric and duodenal ulcer is not difficult. The history of pain coming on two or more hours after food, the periodicity of the attacks, and the intervals of good health, point to the duodenum as the site of the ulcer. After perforation, the point of maximum tenderness usually indicates the site of the ulcer.

In perforation of duodenal ulcer, the mimicry of acute appendicitis may be most marked and perplexing, and frequently it has led to mistakes. The reason of this mimicry is, that in perforated duodenal ulcer the extravasated fluid gravitates to the right iliac fossa, and causes pain, rigidity, and tenderness in this region. To add to the difficulty in

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diagnosis, in some cases of appendicitis there is a history of pain after food. As a rule, however, the interval between the taking of food and the onset of pain is much shorter than is the case in duodenal ulcer. In appendicitis it is rare to get such a clear-cut picture of pain recurring in so definite and ordered a manner as obtains in duodenal ulcer. In both conditions there is tenderness in the right iliac fossa, but in acute appendicitis the rigidity is less marked, and the pain far less agonising than in perforated duodenal ulcer. In acute appendicitis there may be tenderness in the right hypochondrium, but much less marked than is the case in duodenal perforation.

Now that we recognise and are prepared for this pitfall, errors in diagnosis are far less common. In the great majority of cases a correct diagnosis may be made by attention to the following points :

1. The history.

2. The point of maximum tenderness.

3. The temperature—an initial rise in acute appendicitis, and, as a rule, an initial fall in acute perforation of an ulcer.

4. In my experience, in cases of perforated duodenal ulcer, often there is dullness in the right flank, which disappears in part when the patient is turned on his left side. This sign, present on the right but not on the left, is very suggestive of perforated duodenal ulcer.

The Treatment of Acute Perforation of Gastric or Duodenal Ulcers.—There is only one method of treatment for acute perforation, and that is immediate operation. There are on record undoubted instances of recovery without operation. The following case is probably an instance :

An elderly lady, who had suffered from indigestion for years, was sent to me by Dr. Soltau Fenwick. Nine years previously, on the way to Paris, she was seized suddenly with severe

abdominal pain, followed by signs of general peritonitis. She was laid up for many weeks, but eventually recovered. At the operation (nine years later) I had great difficulty in finding the peritoneal cavity owing to extensive adhesions. The upper two feet of the jejunum were matted together by dense adhesions, evidently the result of an old perforation. After a tedious dissection I succeeded in separating the adhesions, and performed gastro-jejunostomy. The patient made an excellent recovery.

Such a favourable result is extremely rare, and is no argument against the rule of immediate operation. No case is too bad for operation, and there must be no waiting for a quiescent period which may never come. Whilst preparations for the operation are in progress, a pint or so of saline should be administered by the rectum, and half a pint in each axilla. By these means, and by the application of plenty of warmth, preferably in the form of radiant heat from electric lamps suspended from a cradle, even a most desperate case may be tided safely through the operation, provided that it is performed with rapidity and gentleness. With regard to the anæsthetic, this should be either gas and oxygen, or ether. I hold very strongly that in these cases, as indeed in all acute abdominal cases, the use of chloroform is absolutely contra-indicated, and prejudices greatly the patient's chance of recovery.

The Technique of Operation for Acute Perforation of Gastric and Duodenal Ulcers.—An incision 1½ inches long is made in the middle line above the umbilicus. If gas or free fluid be found in the peritoneal cavity, the incision is enlarged to three inches. If gastric contents are found distributed generally throughout the peritoneal cavity, I make a stab incision above the pubes at once, through which I pass a large Keith's tube to the bottom of the pelvic cavity. During the remainder of the operation a nurse is instructed

to suck out, at intervals, the fluid in the abdomen by means of a glass syringe attached to a rubber tube of suitable size for being passed into the Keith's tube.

Then a rapid search is made for the perforation. As a rule it will save time if, in a male patient, the duodenum be examined first, in a female patient, the stomach. Often the site of perforation is indicated by a thick deposit of lymph. The perforation is sewn up by two rows of sutures, the first penetrating all coats, the second row being serous sutures. If there be much induration round the perforation, or if the surrounding tissues be very friable, suture of the perforation may be very difficult; but the surgeon must not be satisfied until, by some means, he has effected a complete closure. When complete closure by suture is impracticable, the aperture may be closed by stitching a piece of omentum or small intestine over it, or, as I have done on one occasion, by doubling over a piece of the stomach and stitching it round the edge of the perforation.

Invariably after closure of the perforation I perform a gastro-jejunostomy. I advocated this as a routine measure in my Hunterian lectures delivered in 1906. Briefly my reasons are as follows : As I shall point out later, in dealing with the remote results of operation for perforation, 23 per cent. of the patients who recover from operation relapse subsequently. This in itself is an argument in favour of my practice. But some more urgent reason is required. Our concern is with the immediate saving of life, the ultimate result is a secondary consideration. My contention is that the routine performance of gastro-jejunostomy diminishes the mortality following operation for perforated ulcer. I will indicate briefly the grounds of this belief.

A perusal of hospital records shows clearly that a number of patients die shortly after operation for perforation, either from perforation of a second ulcer, from hæmatemesis, or

from leakage along the line of suture. In a series of 112
consecutive cases I found that 11 per cent. of the patients
died from one or other of these accidents. In a series of
cases published by Dr. H. P. Hawkins, the mortality rate
from these causes was 12 per cent. It will scarcely be
denied that the danger of perforation of a second ulcer and
the risk of hæmorrhage are diminished greatly, if a gastro-
jejunostomy be performed. Further, if the ulcer be situated
near the pylorus, often it is impossible to invaginate the
ulcer thoroughly without causing narrowing of the pyloric
orifice. In his anxiety to avoid this, the surgeon is apt to
turn in a margin round the ulcer insufficient to secure firm
union, with the result that the sutures cut out, and allow
fresh extravasation of gastric contents. If, however, a
gastro-jejunostomy be performed, the ulcer may be enfolded
as freely as may seem desirable ; and further, owing to the
altered physiological conditions produced by the short
circuit, far better conditions are secured to promote healing
of the line of suture.

I think, therefore, that we are justified in regarding as
preventable the deaths which are due to the causes indicated
above ; and the presumption is strong, that a considerable
proportion of these deaths would be prevented by the per-
formance of gastro-jejunostomy. I do not claim a certainty,
only a possibility, but I do urge that a procedure which
holds out even a possibility of reducing the mortality from
perforation by 11 per cent. deserves a very extended trial.

There is, however, an even more weighty reason than
any of the foregoing. There can be little doubt that the
most effective means of draining the peritoneal cavity is
through the intestines and through the intestinal lymphatics.
In order to promote and encourage this drainage, our aim
should be to start intestinal movements as soon as possible
after operation. After gastro-jejunostomy, purgatives may

be given at once with absolute safety, and from experience I am convinced that they act much more certainly and much more quickly when there is a short circuit between the stomach and the jejunum.

This suggestion has been much criticised, but more extended experience only has confirmed me in my belief that it is right, and that the performance of gastro-jejunostomy in all cases, not only improves greatly the patient's immediate chance of recovery, but ensures a more complete and permanent cure.

Two objections have been raised to this procedure : first, that the tissues are not in a condition for a plastic operation, and secondly, that it prolongs the operation unduly.

In my opinion neither objection is valid.   My experience is, that even in cases of septic peritonitis, peritoneal surfaces unite perfectly well, if kept in apposition by the firm pressure of a continuous suture.   To the objection that the performance of gastro-jejunostomy prolongs the operation unduly, my answer is, that it ought not to prolong the operation more than five, or at the outside, seven minutes.   Several times I have sutured a perforated gastric ulcer and performed gastro-jejunostomy in less than twenty-five minutes.

The illustrative case of perforated gastric ulcer which is related earlier in this chapter (p. 186), is a good example of the value of gastro-jejunostomy ; without it I think recovery would have been impossible.

A well-known writer, commenting on my advocacy of a primary gastro-jejunostomy, states that in some very *early* cases a gastro-jejunostomy may be performed primarily, but, that in the majority of cases, it is safer to perform it at a second operation.   He has missed the point of my argument. My chief reason for advocating a primary gastro-jejunostomy is, not to guard against future recurrence, but to improve the patient's immediate chance of recovery.   It is in the later,

and not the earlier cases, that gastro-jejunostomy is indi-
cated most emphatically.

To recapitulate, the performance of gastro-jejunostomy
in addition to suture of a perforated ulcer :

1. Lessens the chance of perforation of a second ulcer in
cases in which more than one ulcer is present.

2. Diminishes the risk of a fatal issue in cases in which
there happens to be two perforated ulcers, only one of which
is discovered.

3. Minimises the risk of hæmatemesis from the ulcer after
operation.

4. Allows more efficient enfolding of the ulcer in cases
in which the ulcer is in the duodenum or in the pyloric third
of the stomach.

5. Promotes more rapid and certain healing of the line
of suture, and so lessens the risk of further leakage.

6. Permits earlier feeding, earlier administration and
more rapid action of purgatives, and so secures more efficient
drainage of the peritoneal cavity.

7. Lastly, but of less importance, it improves the remote
result in those patients who recover.

As regards the toilet of the peritoneum, I hold very
strongly that this is best left to Nature. My belief is that
flushing simply increases shock, and is ineffectual in cleansing
the peritoneal cavity. Neither do I employ mopping or
sponging, as I think it is much more important to get the
patient back to bed as quickly as possible. When once the
perforation has been closed, the peritoneum can deal with
extravasated fluid much better than can the surgeon. The
key to the situation is the promotion of drainage through
the intestinal lymphatics by early intestinal peristalsis.

There is considerable difference of opinion as to the
advisability of drainage. My own view is this : I believe
that drainage does no harm ; I am convinced that in some

cases it does good. Acting, therefore, on the principle, never to take risks which can be avoided, my inclination is in favour of this precaution, neglect of which may do harm, the adoption of which has no injurious result, and may prove of benefit. My usual practice is to put a tube down to the bottom of the pelvis through a stab incision above the pubes, and occasionally in late cases, I leave a drain in the region of the perforation.

As a general rule, at the conclusion of the operation it is advisable to pass a tube down the œsophagus and to wash out the stomach.

Rapidity and gentleness are the main secret of success in these operations. The whole operation, including the performance of gastro-jejunostomy, should be completed in twenty-five, or at the outside, in thirty minutes.

After-treatment.—The patient should be propped up in bed in a sitting position. If there be much shock an injection of pituitary extract (1 c.c. = 17 minims) is administered intramusculary. In all cases continuous saline is given by the rectum for at least forty-eight hours. I never give morphia or strychnine. With a view to draining the intestines and the intestinal lymphatics, I commence the administration of calomel early in these cases. If, at the time of the operation, there be general peritonitis, or an extensive extravasation of gastric contents, the first dose is given as soon as the patient regains consciousness. In less serious cases, the administration of calomel is delayed until the evening or the following morning.

The calomel is prescribed in half-grain doses, repeated every hour or two hours, until three grains or more have been taken. Two hours after the last dose, a seidlitz powder is given, followed an hour or so later by an enema, if necessary. The promotion of early intestinal peristalsis is of the utmost importance, so much so that I am accustomed to

regard the patient as well on the way to recovery, when once there has been a satisfactory evacuation of the bowels.

Feeding is regulated by the condition of the patient. If the patient be in fair condition, and there be much fluid in the peritoneal cavity, nothing is given by the mouth until the bowels have been opened. If, on the other hand, there be little extravasation of gastric contents, or the patient be in bad condition, or very emaciated, I commence the feeding at once, with diluted milk (milk one part, water two parts). In weak, elderly, or feeble patients, usually I give an ounce or two of Sanatogen in milk during the course of twenty-four hours. After the bowels have been opened, soft solids and fluids *ad libitum*, are allowed. When a drainage tube has been used, usually it is left in for a week.

Vomiting is rare after operation, but if it occur the surgeon must be on the watch for acute dilatation of the stomach. If this occur, the stomach should be washed out, and the administration of calomel repeated, followed, if necessary, by a seidlitz powder.

Immediate Results.—Although the results obtained by the surgical treatment of perforation are better than formerly, they are still far from ideal. In 1898 Dr. Keen prophesied that the time was approaching when operations for perforation would be performed within twelve hours, and the mortality would be reduced to 10 per cent. This prediction still awaits fulfilment. In the year 1904 the mortality rate of the cases operated on in twelve of the London hospitals is 48 per cent. Through the courtesy of the surgical registrars of fourteen of the London hospitals, I have been able to ascertain that during the year 1910, 143 operations were performed for perforated gastric ulcer : of these patients 64 died, a mortality rate of 44 per cent. For perforated duodenal ulcer 58 operations were performed : of these patients 23 died, a mortality rate of 39 per cent.

Thus so recently as the year 1910 the percentage of deaths following operation for perforated gastric and duodenal ulcers was still as high as 43 per cent.

Many writers have laid stress on the importance of early operation. The tables published by Weir and Foote, and Keen and Tinker, have been quoted extensively as showing that the cases of early operation have a much higher percentage of recoveries than have those operated on later. Thus in Weir and Foote's series the mortality of those cases operated on within twelve hours is 39 per cent., and 76 per cent. in operations performed between twelve and twenty-four hours after perforation. In Keen and Tinker's tables of seventy-eight cases, the mortality of those operated on within twelve hours is 19 per cent., and of those operated on within from twelve to twenty-four hours 50 per cent. Keen and Tinker also divide the cases in their table into two periods, those before the year 1896 and those after that year. In the first period the mortality of those operated on within twelve hours is 28·5 per cent., in the second period it is only 16·6 per cent. " This rapid fall," remarks Dr. Keen, " in the mortality of the cases operated on within the same interval after perforation shows that our technique, and therefore our results, are improving."

These tables, however, do not justify this sanguine conclusion, for they are not consecutive, but isolated, published cases, and therefore comprise an undue proportion of successes, for of the 78 cases, 52—that is, 66 per cent.—of the patients recovered. In the series of consecutive cases referred to above (p. 182), the mortality rate, even in those operated on within twelve hours, is 47 per cent. ; in those operated on within from twelve to twenty-four hours, 53 per cent. ; in those operated on within from twenty-four to forty-eight hours, 83 per cent. ; and in those operated on over forty-eight hours after perforation, 50 per cent. So that in the

consecutive series of cases, early operation does not seem to have quite the importance which Keen and Tinker's table appears to show. I need hardly say, I do not suggest that operation should be delayed. Undoubtedly, the earlier operation is performed, the better. The figures which I have given, show the untrustworthiness of statistics compiled from isolated cases, and also that other factors besides early operation may have an important bearing on the result, and of these probably the amount and nature of the extravasation, the presence of other ulcers, and the rapidity and completeness with which the operation is performed, are the most important. But while admitting this, we must not lose sight of the axiom, that perforated gastric or duodenal ulcer is a catastrophe demanding early recognition and immediate operation.

Remote Results.—In view of the suggested advisability of performing gastro-jejunostomy it is important to ascertain what proportion of the patients operated on for perforations suffer subsequently from gastric troubles. Numerous isolated instances of relapse have been recorded.

This subject is one which merits full investigation by inquiry into the after-history of a large number of patients in whom perforated ulcers have been sutured. Of 54 patients in my series who recovered after operation for perforated gastric or duodenal ulcer, I have traced 32. All those in whom gastro-jejunostomy was performed in addition to suture of the ulcer, have remained quite well, with the exception of one patient who died from spinal meningitis. Of the remainder, 17 have suffered from gastric trouble. Of these 17 patients, one died from a second perforation, two subsequently required gastro-jejunostomy, while at the time of inquiry nine had symptoms suggestive of ulcer, and five had symptoms due probably to simple dyspepsia. Therefore, if we regard these figures in the most favourable light,

by assuming that all the untraced patients have remained quite well, we see that of the cases in which gastro-jejunostomy was not performed 12 patients—that is, nearly 23 per cent. of the patients who recovered—have had symptoms suggesting a recurrence of ulcer, one case proving fatal, and two patients requiring further operation. The ultimate results in the cases comprising the series published by Dr. Hawkins, to which I have alluded, are almost identical. No less than 23 per cent. of the patients sought relief at the hospital for gastric trouble after their operation.

A study, therefore, of the after-histories of patients operated on for perforated gastric or duodenal ulcer, appears to me to furnish most convincing evidence in favour of my contention, that gastro-jejunostomy as well as suture of the ulcer, is indicated in the treatment of perforated gastric and duodenal ulcers.

Chronic Perforation.—In chronic perforation the ulcer destroys the gastric wall slowly, while plastic peritonitis at the base of the ulcer results simultaneously in adhesion of the stomach to surrounding parts. Thus, when the base of the ulcer is destroyed, the escape of the gastric contents is limited by the adhesions which have been formed. Chronic perforation of a gastric ulcer is more common in the posterior wall of the stomach, and frequently the ulcer erodes the pancreas. If the adhesions yield, gastric contents may leak slowly into the surrounding adhesions, leading to a peri-gastric abscess. The abscess thus formed may increase slowly, in size, and finally rupture into a hollow viscus, or track upwards (sub-phrenic-abscess) and perforate the diaphragm into the pleural or the pericardial cavity. A sub-phrenic abscess may be anterior or posterior. An anterior abscess results from perforation of an ulcer of the anterior gastric wall. The pus tracks upwards between the right lobe of the liver and the diaphragm to the right of the falciform ligament,

or between the left lobe and the diaphragm to the left of the falciform ligament. In some instances the abscess may destroy the overlying skin by pressure, and so discharge externally, but more usually perforation of the diaphragm ensues. The lower limit of the abscess is formed by dense adhesions, the result of plastic peritonitis.

A posterior abscess is limited at first, by the boundaries of the lesser sac, and extends up to the diaphragm over the posterior and upper surfaces of the liver, while later, as in the case of an anterior abscess, perforation of the diaphragm may occur. It must be borne in mind, that a sub-phrenic abscess may result from imperfect drainage of the peritoneal cavity after operation for acute perforation.

Sub-phrenic abscess from perforation of the stomach, or duodenum, is far more common on the left side than on the right.

Symptoms and Diagnosis of Perigastric Abscess.—The symptoms of perigastric abscess may be very indefinite, the signs obscure, and consequently the diagnosis of some difficulty. In some cases there is a history of a sudden onset of pain, indicating the time at which perforation occurred. In others this suggestive feature is absent. The chief symptoms are those of septic absorption, daily rise of temperature, night sweats, occasional vomiting, and later, attacks of coughing accompanied by dyspnœa. As the abscess increases in size there may be physical signs of its presence—impaired movements and bulging of the lower part of the chest, and displacement of the heart. The sudden rupture of the abscess into a hollow viscus, or through the diaphragm into the pleura, may be attended with severe collapse.

The following case illustrates these features very well :

A man, aged twenty-six, suffering from a gastric ulcer, while asleep in hospital, was seized suddenly, during the night, with

severe abdominal pain. A few hours later I opened the abdomen and found a very extensive perforated gastric ulcer of the anterior wall of the stomach at the extreme cardiac end. I sutured the perforation and performed gastro-jejunostomy. The patient was desperately ill; a large area round the abdominal wound sloughed, exposing the intestines.

The temperature remained fairly regular until three weeks after the operation, when he had a sudden rise to 103° F.

Gradually his temperature fell again, and became normal in a few days. A week later he had another sudden rise. During the next three weeks he had a hectic temperature varying between 98° F. and 103·8° F., with occasional vomiting. A sub-phrenic abscess was suspected, and he was explored on three occasions, but no pus was discovered. A few days later he had a severe rigor, and after this the daily variations in temperature were less great. One week later he had a sudden attack of severe dyspnœa, accompanied by extreme collapse, and a pulse-rate of 140. The whole of the left side of the chest was dull to percussion. He was aspirated, and 63 ounces of pus were drawn off. After being desperately ill for a few days he improved slowly; the temperature varied between 98° F. and 100° F.

One month later he was aspirated again, and 40 ounces of pus withdrawn. Four days later a rib was resected, and a drainage tube inserted. Thereafter he improved steadily, and was discharged with a sinus at the site of resection, after having been seven months in hospital. The sinus persisted for nearly a year after his discharge, and I suggested that he should come in and have a further resection of ribs. The mere suggestion of an operation was sufficient, as within a month the sinus was soundly healed. Seven years later the man was in perfect health and suffered no inconvenience from weakness of the abdominal wall, although over an area of three inches above the umbilicus this consisted of skin only.

The Treatment of Sub-phrenic Abscess.—Sub-phrenic abscess is inevitably fatal, unless it be treated surgically.

As a rule a sub-phrenic abscess is best opened and drained through the pleura after resection of one or more ribs. An incision about four inches long is made over the ninth rib on the right side, or over the seventh on the left side, and about three or four inches of rib resected. If the pleura do not contain fluid, the diaphragm and both layers of pleura are stitched to the chest wall. If the ribs be close together it may be necessary to resect portions of two ribs.

The edges of the wound are protected with gauze, and a director is pushed through the diaphragm into the abscess cavity. The opening is stretched with a pair of forceps and a large drainage tube inserted. It is not advisable to irrigate the abscess cavity either at the time of operation or subsequently. When a sub-phrenic abscess bulges in the epigastrium, or below the costal margin, it may be possible to open it by an incision in the epigastrium, or parallel with the costal margin. Sometimes a counter-opening in the loin may be made with advantage.

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* In this paper Dr. Deaver supports strongly the view expressed in this chapter, that a primary gastro-jejunostomy is advisable.

CHAPTER XII

THE COMPLICATIONS OF GASTRIC AND DUODENAL ULCERS—*continued*

2. HÆMORRHAGE

Gastrorrhagia or Gastric Hæmorrhage is made evident by hæmatemesis or melæna, or by both. It is a symptom both of acute and of chronic ulcer.

In a number of cases in which operation has been undertaken for gastric hæmorrhage, no definite ulcer has been found, but examination of the interior of the stomach has revealed a number of distinct and separate bleeding-points. More than thirty years ago Murchison reported two cases of fatal hæmorrhage from minute ulcers, which had perforated a small artery in the stomach wall. He observes that " they are remarkable for the minuteness of the ulcers, which are little more than hæmorrhagic erosions, but also for the absence of the usual symptoms of ulceration of the stomach. Neither of the patients suffered from vomiting prior to the occurrence of the hæmorrhage." Chiari, in 1882, described a similar case. More recently, Dieulafoy recorded a considerable number, and divided them into two groups—simple erosions and exulceratio simplex. The simple erosions appear to be mere abrasions of the gastric epithelium. In such cases the gastric mucous membrane, when examined during life, appears to be studded with minute bleeding-points, probably the initial stage of the ordinary round ulcer of Cruveilhier. In exulceratio simplex there is a greater

destruction of tissue, so that the minute arteries beneath the muscularis mucosæ are exposed.

Both varieties may give rise to alarming hæmorrhage. I have seen another variety which, so far as I know, has not been described. On opening a stomach subsequent to an extremely severe attack of hæmatemesis, a raised, vividly red patch, of the size of half a crown, was observed. No loss of substance was apparent, and the surface was oozing blood, seemingly from the acuteness of the inflammation. The wound was sewn up again and the patient had no further trouble, so that in the light of the after-history the operation was unnecessary.

Contrary to the accepted teaching, I believe that in many cases of duodenal ulcer, the hæmorrhage comes, not from the ulcer as is generally supposed, but from the mucous membrane of the stomach. In several of my cases of duodenal ulcer, microscopic sections of the gastric mucosa show blood escaping between the epithelial cells. Probably the same holds good in some cases of gastric ulcer.

Hæmatemesis cannot be regarded as pathognomonic of gastric ulcer. Not infrequently it occurs in cases of chronic appendicular disease, and is the effect of the hyper-secretion which, as I have shown elsewhere, often results from chronic appendicitis. Hæmorrhage from the stomach may also be secondary to cirrhosis of the liver, or due to rupture of varicose veins in the gastric mucosa, or to rupture of an aneurysm.

Since Dr. Soltau Fenwick pointed out the clinical and pathological distinction between acute and chronic ulcers, most clinicians are agreed that hæmatemesis due to an acute ulcer presents a totally different clinical picture from the hæmatemesis which results from a chronic gastric ulcer, and this difference has an important bearing on the surgical treatment of the bleeding. The pathology of the bleeding

in acute ulcer and simple erosion is so similar, that surgically
we need not draw any distinction between these two varieties.

In acute ulcer, according to Dr. Fenwick, severe hæmate-
mesis is the first indication of the disease in 75 per cent. of
the cases. Usually the hæmorrhage is due to erosion of a
small branch of the coronary artery, and is sudden and
profuse, so that as much as a pint, or more may be lost in
a few minutes. As a rule, Nature's means for arresting
hæmorrhage, namely, the contraction of the artery, aided
by the diminution of the heart's action due to loss of blood,
are sufficient to stay the bleeding. The artery becomes
occluded by the formation of an internal and external clot
and the hæmorrhage rarely is repeated.

In hæmorrhage from a chronic ulcer, on the other hand,
there is almost invariably a history of chronic dyspepsia for
months or years. The amount of bleeding varies from a
minute quantity, only detected by an examination of the
fæces for occult blood, to an amount so great as to lead
to an immediately fatal issue (*l'hémorrhagie foudroyante*),
although fortunately such an event is rare. As a rule the
hæmorrhage is less copious and less alarming than that from
an acute ulcer.   There is also this important difference :
the hæmorrhage from an acute ulcer once arrested is seldom
repeated, the hæmorrhage from a chronic ulcer tends to
recur after a quiescent interval, until the patient is reduced
to a condition of profound anæmia by repeated hæmorrhages.
It is the repetition of the hæmorrhage from a chronic ulcer,
which is so characteristic, and of such serious import.

What is the reason of this difference ? In acute ulcer
there is little or no surrounding induration, the arteries are
not thickened by inflammatory infiltration, the tissues are
soft and yielding, and consequently Nature is able to use
her own means for the arrest of hæmorrhage.

In a chronic ulcer the eroded artery is thickened by

chronic inflammation, and lies in rigid, indurated tissues, and so cannot close the opening in its wall by contraction. The formation of a clot is sufficient to arrest the bleeding temporarily, but the clot, exposed to the gastric juice, and to the discharge from the ulcer, becomes disintegrated, and leads to the recurrence of the hæmorrhage.

The Surgical Treatment of Gastrorrhagia.—The first suggestion of surgical intervention for gastric hæmorrhage was made by Rydygier in the year 1882. His suggestion, like many other original propositions, was received with coldness, and it was not until five years later that Mikulicz performed the first operation for hæmorrhage from a gastric ulcer, while it was reserved for Professor Roux of Lausanne, whose work I have had the privilege of witnessing, to have the credit of performing the first successful operation of this nature. In this case the source of bleeding was the coronary artery, and he excised the ulcer and ligatured the artery on both sides of the bleeding-point.

There is a considerable difference of opinion as to when to interfere in cases of gastric hæmorrhage. At present few are prepared to agree with Dieulafoy's opinion, that operation should be performed *during* the first bleeding, if as much as half a litre of blood be lost, or if, after a loss of a smaller quantity, the hæmorrhage be repeated within twenty-four hours. Dieulafoy believes that a third attack is likely to prove fatal, a view which, though not generally accepted, is supported in some measure by the facts recorded by Savariaud.

Granted, however, that surgical measures sometimes are indicated, the questions arise, **In what class of case should we operate? When should we operate? and How should we operate?**

1. **In what Variety of Gastric Hæmorrhage is Operation ndicated?**—By recognising the distinction between bleeding

from an acute ulcer or from a simple erosion, and bleeding from a chronic ulcer, we can form fairly clear indications for surgical treatment.

Pathologically, we may classify hæmorrhage from an acute ulcer as a primary hæmorrhage, and bleeding from chronic ulcer as a form of secondary hæmorrhage, and we can apply some of the principles which guide us in the treatment of primary and secondary hæmorrhage in other regions of the body. The first general principle in the treatment of primary hæmorrhage is, that no operation should be performed on an artery unless it bleeds. Therefore, in hæmorrhage from an acute ulcer, when once the bleeding has ceased, whether spontaneously or by the aid of medical art, we should hesitate to interfere. There is a reasonable expectation that the bleeding vessel will become securely occluded. If, in spite of absolute rest, the hæmorrhage start anew, we may conclude that Nature has failed, and if the second hæmorrhage has been a severe one, surgery must step in. *But* (and this is an important point) absolute rest must have been tried and have failed. I emphasize this, because in several of the cases I have seen, the second attack of hæmorrhage apparently has been recurrent or reactionary, due to displacement of the clot from too early stimulation, or injudicious moving of the patient. This also has been the case in some, at least, of the recorded cases. In these circumstances we may give Nature one more chance before deciding on operation. In bleeding from a chronic ulcer our guide must be the first general principle laid down for the treatment of secondary hæmorrhage—namely, that even if the bleeding have ceased, an operation is necessary to prevent its recurrence. Of course the degree of bleeding must be taken into account. Just as in slight secondary hæmorrhage elsewhere, we should first try simpler means ; so an operation is not indicated necessarily after every slight hæmorrhage

from a chronic ulcer. We must remember, however, that in chronic ulcer, it is probably not mechanical displacement, but septic disintegration, which starts the hæmorrhage afresh, so it is probable that the bleeding will be repeated.

Therefore, if already the patient have lost a considerable amount of blood, an operation is advisable to prevent the serious and possibly fatal result of further loss. Likewise, if the hæmorrhage, although slight, be frequently repeated, the indication for operation is clear. The risk of operation, if undertaken during a quiescent interval, probably is less than the risk from further hæmorrhage, and the patient gains a double advantage, prevention of further hæmorrhage and cure of the gastric ulcer.

2. **When should we Operate?**—Mikulicz took a very pessimistic view of the results of operation during the progress of acute hæmorrhage. He operated on four patients with only one recovery. Billroth had no better results. In the discussion on Mikulicz's paper, Korte gave similar testimony.

In spite of isolated successes, the weight of evidence seems against operation during the progress of the hæmorrhage ; the collapse induced by the loss of blood is favourable for the arrest of hæmorrhage, but unfavourable for operation, and there is not, as in perforation, the risk of increasing peritonitis through delay. Further, in by far the greater number of instances, it is not the initial hæmorrhage which causes death, but the repetition of it before the patient has recovered from the resulting anæmia. It is always dangerous to lay down hard and fast rules. To do so may give those who come after us cause " to reflect on the vanity of received opinion," but both from the cases I have seen, and from a study of the recorded cases, I think it is open to grave doubt, whether it is ever expedient to operate during the progress of gastric hæmorrhage. The number of cases in

which the hæmorrhage is fatal immediately is very small (3 to 5 per cent.), and the percentage mortality of such cases is less than the mortality from operation during gastric hæmorrhage. There are quite a number of cases on record (and I have seen three myself) in which the stomach has been opened for acute hæmorrhage without the discovery of any definite source of bleeding. The stomach has been sewn up and the patient has recovered. Operation has thus been an unnecessary complication to the patient's convalescence. Such cases emphasize the advisability of trusting to Nature more, and resorting to surgery less, in the treatment of acute gastric hæmorrhage.

3. **How should we Operate?**—Many different procedures have been adopted. Mikulicz, in the four cases to which I have alluded already, applied the thermocautery to the base of the ulcer. Three of the patients died within twenty-four or forty-eight hours. The same method was employed by Korte, but the hæmorrhage continued until the death of the patient eight days later. In other cases the ulcer has been excised, ligatured *en masse*, or packed with iodoform gauze, and in others gastro-jejunostomy has been performed. In cases in which no definite ulcer has been found, the bleeding-points have been picked up and ligatured *en masse*.

If we can rely on gastro-jejunostomy to prevent recurrence of the hæmorrhage, it is undoubtedly the simplest procedure to adopt, and we have good evidence that an efficient short circuit between the stomach and jejunum *is* an effective method of treating gastric hæmorrhage. Mr. Mansell Moullin advocates dealing with the bleeding-point, either by ligature *en masse*, or by excision of the ulcer. He argues in support of his view that there are at least two cases on record, in which bleeding has persisted in spite of gastro-jejunostomy, and he himself has had a case in which one attack of hæmatemesis followed this operation. That some-

times hæmorrhage does recur after gastro-jejunostomy, is not open to question, but such cases are rare, and I believe that the majority of them come under one of four categories. First, those in which malignant disease exists ; secondly, those in which a mechanical appliance has been used to effect the anastomosis ; thirdly, those in which the anastomotic opening has become, or originally was, inefficient ; fourthly, those in which hæmorrhage occurs from the mucous membrane in the line of suture.

In the museum of St. Bartholomew's Hospital is a specimen (No. 1951C), which appears to show that a fatal hæmorrhage may ensue from the granulating area caused by the pressure of the button on the tissue surrounding the opening.

Some years ago I was present at the autopsy on a man who had died from hæmatemesis fourteen days after gastro-jejunostomy. I was able to demonstrate that water injected into the stomach did not escape through the efferent opening, and I have seen two other cases in which a similar condition obtained. In some of the recorded cases it is stated that regurgitant vomiting accompanied the hæmorrhage, a fact suggesting obstruction of the anastomotic opening. That with a distended stomach hæmorrhage should recur, is not surprising.

I believe that in the majority of cases in which hæmorrhage occurs after gastro-jejunostomy, the bleeding is not from the ulcer but from the cut mucous membrane round the anastomosis. There is a specimen in the St. Bartholomew's Hospital Museum (No. 1951a₃) from a patient who died from hæmorrhage after gastro-jejunostomy, in which it is clear that the bleeding was from the anastomosis.

Such cases do not prove that the principle of treatment by gastro-jejunostomy is wrong, but that the method of carrying it out is faulty. Probably the cases are very few

in which hæmorrhage from the ulcer has recurred after gastro-jejunostomy. Excision is a more serious operation, especially in a patient suffering from the effects of loss of blood, and even after this method of treatment, recurrence of the hæmorrhage is not unknown (see Plate XL, Fig. 57).

The old rule of surgery, to cut down upon and to tie the bleeding vessel, requires modification in the light of modern experience. That we must look our enemy in the face, is an aphorism which sounds well, but one which may lead to injudicious surgery, if applied in every case. The indirect method of treatment by gastro-jejunostomy is simpler, and the proportion of failures is exceedingly small. There is no doubt, that in many instances, excision of the ulcer does effect a cure, but it is not readily applicable when the ulcer is on the posterior wall of the stomach, the situation of most ulcers which bleed. Further, it may prove ineffectual when a second bleeding, but undiscovered, ulcer exists. Ligature of the bleeding-points *en masse*, while satisfactory in many instances, appears to be less dependable than gastro-jejunostomy.

Supposing, that after opening the stomach, the bleeding-point be not discovered, what is to be done ? Clearly, if the hæmorrhage has been repeated in such quantity, or so frequently, that an operation is justifiable, gastrotomy alone is not likely to prevent its return. If the hæmorrhage be such as to necessitate operation, the probability is that it will recur if nothing more than an exploratory operation be performed. Under such circumstances it appears to me to be better practice to perform gastro-jejunostomy at once, without opening the stomach to search for the bleeding-point.

Gastro-jejunostomy possesses these advantages : that it is applicable equally to cases of acute, or chronic ulcer, it renders exploration of the stomach unnecessary, and it can be

performed in very short time. Further, having decided that operation is necessary, we start with a definite procedure in view, whereas an attempt to discover the exact source of bleeding may lead to a prolonged operation on a patient little able to bear it. Human nature being what it is, it is difficult to stay our hand, if we do not discover at once the source of the bleeding which we seek. The rules which I have formulated for my own guidance in these difficult cases are as follows :

In hæmorrhage from an acute ulcer, erosion, or exulce-ratio simplex—that is, when the onset of bleeding is sudden and history of previous gastric ulcer is absent—first give a thorough trial to absolute rest in bed, ice applied to the abdomen, Trippier's hot-water injections by the rectum, and avoidance of food by the mouth for at least four or five days. If a second profuse hæmorrhage occur, perform gastro-jejunostomy, provided it be quite clear that the second hæmorrhage has not been brought on by want of absolute rest, by too early stimulation, or by too early administration of food. It is advisable to delay operation until the patient has recovered from the collapse induced by the hæmorrhage. In hæmorrhage from a chronic ulcer—that is, in cases with a definite history of pain after food, vomiting, and possibly previous slighter attacks, of hæmatemesis—perform gastro-jejunostomy after one severe attack, or after several slighter attacks of hæmatemesis, if the loss of blood be causing serious anæmia.

As I have indicated previously (p. 209), my belief is that in duodenal ulcer, most commonly the hæmorrhage comes, not from the ulcer, but from the mucosa of the stomach. The principles of treatment are the same as in hæmorrhage from a gastric ulcer, and the method of applying them identical. There is, therefore, no necessity to add anything to what is contained in the preceding paragraphs.

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CHAPTER XIII

THE COMPLICATIONS OF GASTRIC AND DUODENAL ULCERS—*continued*

3. CHRONIC DILATATION OF THE STOMACH

Chronic dilatation of the stomach may arise from gastric or duodenal ulcer in two ways: **(i)** cicatrisation of an ulcer situated at, or near, the pylorus may cause definite mechanical obstruction to the exit of the gastric contents, or **(ii)** the presence of an ulcer near the pylorus may cause spasm of the pylorus, which may persist even after the ulcer has healed.

During the process of healing, a gastric ulcer, like any other ulcer, contracts. This contraction causes certain changes in the stomach which lead to well-marked symptoms, depending on the situation of the cicatrix. If the ulcer be at, or near the pylorus, cicatrisation may produce pyloric stenosis, while if the ulcer be in the body of the stomach, the constriction produced by the cicatrisation of the ulcer may divide the stomach into two portions, communicating by means of a narrow isthmus—a condition known as "hour-glass stomach."

Pyloric stenosis may be caused also by pressure from without, as, for example, by the pressure of a tumour growing from the liver or pancreas, or by inflammation of a neighbouring organ, as when an inflamed gall-bladder becomes adherent to the duodenum, with the result that the duodenum also becomes infiltrated with inflammatory products.

The symptoms of pyloric obstruction, dilatation of the stomach, vomiting, and peristaltic movement of the stomach visible through the abdominal wall, form a clinical feature

which is recognised readily. The nature of the vomiting is characteristic. It does not occur after meals, nor necessarily every day. After food there is a sense of discomfort which increases gradually with each succeeding meal, until at last relief is obtained by vomiting. Sometimes the quantity brought up is enormous, and the vomit consists of fermented food mixed with much mucus. Often articles of food taken some days previously are found in the vomit.

The extent of the vomiting does not depend entirely upon the degree of narrowing of the lumen of the pylorus. I have seen profuse vomiting with a pylorus having quite a fair lumen. I think the important factor is the extent of the interference with the contraction of the circular fibres, by the formation of fibrous tissue. When the musculature of the pylorus is replaced by fibrous tissue, the wave of peristaltic contraction, originating in the stomach, is arrested at the pylorus, and so the emptying of the stomach is imperfect.

The outline of the dilated stomach usually is plainly visible through the abdominal walls, and waves of peristalsis may be seen and felt passing across the upper half of the abdomen from left to right. The extent of the dilatation can be ascertained by palpation and percussion, after distending the stomach with air.

The most important diagnostic sign of pyloric stenosis is the presence of food in the stomach in the early morning. If 340 c.c. (12 oz.) of milk be given overnight, on passing the stomach tube ten hours later, 200 c.c. or more of fluid containing food may be recovered. If some raisins or other easily recognisable food be given with the milk, some of them will be found in the stomach next day.

If the stomach be empty in the morning we can be certain that pyloric stenosis is not present.

The treatment of organic pyloric stenosis is gastro-jejunostomy.

CHAPTER XIV

THE COMPLICATIONS OF GASTRIC AND DUODENAL ULCERS—*continued*

4. HOUR-GLASS STOMACH

By the term "hour-glass" stomach is meant a stomach which is divided into two cavities by a constriction. It is a serious complication of gastric ulcer. Not infrequently the patients suffering from it are so emaciated, that they are supposed to have advanced malignant disease, a diagnosis supported by the gastric findings, which resemble closely those of gastric carcinoma. The patient, therefore, may be considered beyond the reach of surgery, and so is allowed to die unrelieved. Such an error is one to be guarded against, as there are few conditions in which relief is so immediate, and so complete, as it is by the surgical treatment of hour-glass stomach.

The first accurate description of the condition was given by Morgagni. Usually the constriction is in the middle third of the stomach, sometimes near the pylorus, or more rarely, near the cardiac end. In most of the specimens which I have examined, the communication between the two pouches has been smaller than the external appearance of the stomach would suggest. Indeed, it is of practical importance to recognise, that on opening the abdomen, an examination of the external surface of the stomach may give little indication of the serious constriction within. In one patient upon whom I operated, there was only a slight puckering on the anterior wall of the stomach, but internally,

an annular stricture so tight, that it admitted only the tip of my little finger.   (See Plate XLII, Figs. 60 and 61.)

In the majority of the specimens and recorded cases, the greater curvature is drawn up towards the lesser, but in one case figured by Schwarz, the lesser curvature is drawn down towards the greater.

The Pathology of Hour-glass Stomach.—Two varieties of hour-glass stomach have been described—the congenital and the acquired; but Mayo Robson, and Moynihan have expressed a doubt as to the existence of hour-glass stomach as a congenital deformity.   A perusal of the recorded cases, and an examination of the specimens in the London museums, have led me to a similar conclusion.   The cases brought forward by Carrington have been accepted generally as undoubted examples of congenital deformity, but in all of them there were either ulcers, or scars of previous ulcers, or adhesions.   The presence of ulcers or scars was noted also in the cases recorded by Roger Williams, Hirsch, and Hudson.   Sievers records a case of " congenital " hour-glass stomach in a woman, aged thirty-eight years, who died from suppurative peritonitis due to perforation of a gastric ulcer in the pyloric pouch.   No macroscopic or microscopic evidence of pathological change could be discovered in the constricted area.   A very small ulcer, however, may lead to very extensive cicatrisation, and the scar of an ulcer is so difficult to detect, hidden as it may be in folds of the mucous membrane, that unless sections of every portion of the constriction be examined, the scar of a healed ulcer may be overlooked easily.   (See Plate XLI, Fig. 58, p. 182.)

As regards Hudson's hypothesis, that the ulceration in these cases is secondary, it is at least suggestive, that in Sievers' case the ulcer which perforated was on the distal side of the constriction and not in the cardiac pouch, where the stagnation and irritation of food must have been greater.

PLATE XLII.
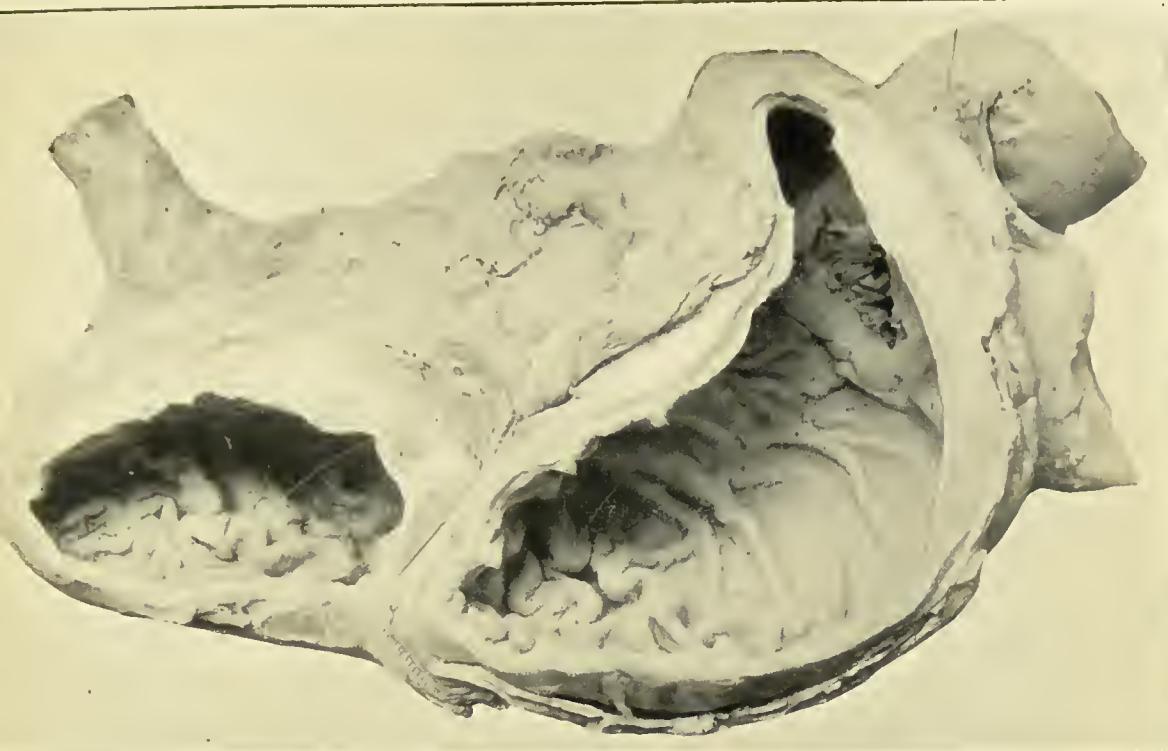
FIG. 60.—The interior of an hour-glass stomach viewed from behind.
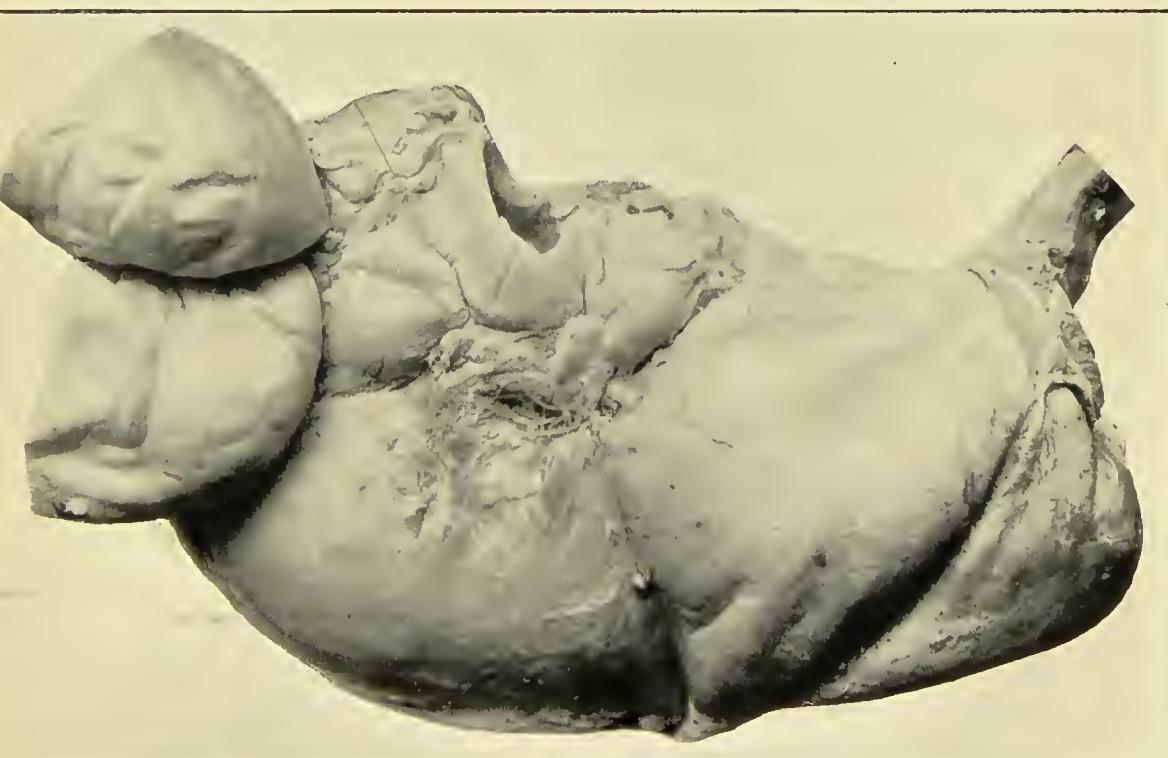
FIG. 61.—The front view of the exterior surface of the specimen shown in preceding figure. Note how little evidence there is of the marked constriction which exists within.

It is a striking fact, that in all the cases which I have been able to discover, symptoms were absent during early life. Thus, in Sievers' case the patient was quite well until the age of twenty-six years, and in Hirsch's case gastric symptoms began at the age of sixteen years, and increased gradually after the age of forty years, so that during the last ten years of her life the patient was unable to take solid food. The absence of symptoms in early life is difficult to reconcile with the hypothesis of a congenital origin, while the appearance of symptoms between the ages of fifteen and thirty years is strongly suggestive that gastric ulcer is the causal factor. It is, too, most significant, that up to the present time, so far as I can discover, no instance of an hour-glass stomach in a fœtus has been recorded.[1] Possibly the following observation may explain some of the specimens of so-called congenital cases. I was present at a post-mortem examination when an apparently typical specimen of an hour-glass stomach was discovered. Through an oversight the specimen was not put into formalin at once. On examination of the specimen next day, I found that all trace of the constriction had vanished, and, presumably, what had appeared to be constriction was really only a muscular contraction which had persisted for some time after death. Had the specimen been placed in some hardening reagent immediately, doubtless it would have been preserved as a typical example of congenital hour-glass stomach.

The cases of Hochenegg, Doyen, and Cumston are described as congenital, but in all of them there was evidence of ulceration. In Doyen's case the stomach had become

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[1] Sandifort (*Observationes anatomico-pathologicae*, published 1779, liber iii., p. 11) figures a stomach with a slight narrowing midway between the cardiac and pyloric ends, which was obtained from a fœtus. The narrowing is very slight, and in the absence of any examination of the interior of the organ, it can hardly be recognised as an hour-glass stomach.

adherent to the anterior abdominal wall, and on separation of the adhesions a perforation of the stomach was discovered.

In some instances the stomach is divided by two constrictions into three pouches, forming the so-called " trifid stomach." Of this variety I have had two examples.

Hour-glass stomach may be the result of :

(i) Cicatrisation of a gastric ulcer.

(ii) Puckering due to perigastric adhesions.

(iii) Adhesions of the stomach to the anterior abdominal wall following perforation of a gastric ulcer.

(iv) Contraction of a gastric carcinoma.

The Symptoms and Signs of Hour-glass Stomach.—The most constant symptom of hour-glass stomach is pain. In all of my cases pain has been marked, and its presence is noted in 93 per cent. of the recorded cases. Vomiting is present in 85 per cent. of the cases, while gastrorrhagia as evidenced by hæmatemesis or melæna, or by both, occurred in 31 per cent. of the recorded cases. In one of my cases the hæmorrhage was so severe as to prove almost fatal.

Usually, the onset of pain does not follow the ingestion of food immediately, but after an interval varying from half an hour to two hours. In the later stages of the disease the pain may be constant. It is " boring " in character, and is referred to the epigastrium, occasionally with radiation to the back. In the early stages of the disease heartburn (acid eructations) often is one of the symptoms.

The signs are those of dilated stomach which, in many of the cases, has been attributed to pyloric stenosis. If, however, the condition be suspected, there are certain signs which usually will enable the observer to make a correct diagnosis. The signs are :

1. If a definite quantity of water be introduced into the stomach by a tube, a portion of the fluid escapes into the

pyloric pouch, so that only the fluid remaining in the cardiac pouch can be recovered. Wölfler, who called attention to this sign, said that " Some of the fluid introduced seemed to disappear as though it had flowed through a large hole."

2. If the stomach be washed out until the returned fluid is clear, after waiting a few minutes there may be a gush of dirty, offensive fluid, which has regurgitated from the pyloric into the cardiac pouch (Wölfler's second sign).

3. The stomach resonance is percussed carefully, and then a seidlitz powder is given in two separate halves. After an interval of thirty seconds, there is considerable increase in the resonance of the upper part of the stomach, while the lower part remains unaltered. If the abdomen be watched for a few seconds, the pyloric pouch sometimes may be observed to fill gradually and become prominent· (Moynihan's sign).

Personally, I distend the stomach with air by means of an inflater, in preference to the use of the seidlitz powder.

4. Sometimes after apparent emptying of the stomach by means of a tube, there is still a splashing sound on palpating the upper part of the abdomen. This sign is known as " paradoxical contraction." The splashing sound is produced in the pyloric pouch, which, of course, is undrained by the passage of the stomach tube into the cardiac pouch.

5. Usually examination of the stomach by the X-rays, after administration of a bismuth meal, will show clearly the presence of an hour-glass stomach. This is a valuable positive sign ; too much reliance must not be placed upon it if it be negative.

Of all these signs the most valuable are, the washing out test and the distension test (Nos. 1 and 3).

Gastric Analysis.—The gastric findings are those of chronic gastritis. I have never found any free hydrochloric acid in a case of hour-glass stomach. I have not seen this

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mentioned by previous writers. It is an important truth, as the absence of free hydrochloric acid leads frequently to a diagnosis of malignant disease.

At first sight the absence of free hydrochloric acid is difficult to explain. The oxyntic cells are in the cardiac end of the viscus, and therefore we should expect that free hydrochloric acid would be secreted as usual. Possibly the explanation is that the gastric hormone, which is secreted in the pyloric pouch, does not reach the cardiac end of the stomach, and so the oxyntic cells are not stimulated into activity.

The Diagnosis of Hour-glass Stomach.—When the constriction is in the pyloric half of the stomach, the dilated cardiac pouch is often mistaken for a dilatation of the whole stomach due to pyloric stenosis.

Usually Wölfler's two signs, already mentioned, supplemented by distension with air, will enable a correct diagnosis to be made. When the constriction is near the cardiac end of the stomach a correct diagnosis is less easy. The cardiac pouch may be so small that there is difficulty in swallowing, and also regurgitation of food, very soon after it is taken. Such symptoms suggest œsophageal obstruction. The passage of an œsophageal bougie will exclude this condition, if the bougie can be passed more than sixteen inches from the teeth.

Many of the patients are so emaciated and cachectic as to suggest gastric carcinoma. A long history and severe pain suggest inflammatory rather than malignant disease, and in my experience the total acidity in hour-glass stomach is not so low as it is in a case of carcinoma of such long standing.

In hour-glass stomach of inflammatory origin usually the total acidity is about forty or fifty, while if the disease be malignant we may expect a total acidity of twenty to thirty, or even less.

The Treatment of Hour-glass Stomach.—The following procedures may be adopted in the surgical treatment of hour-glass stomach :

1. Gastro-jejunostomy.
2. Gastroplasty.
3. Gastro-gastrostomy.
4. Stretching the constriction.
5. Partial gastrectomy.

In addition to one or more of these operations, it may be necessary also to divide gastric adhesions (gastrolysis).

In operating for hour-glass stomach it is most important to examine carefully the whole stomach before deciding what is to be done, otherwise the presence of a third pouch, or of pyloric stenosis, may be overlooked.

Gastroplasty is the simplest method of dealing with hour-glass contraction, but it is open to the same objection as pyloroplasty for pyloric stenosis—namely, the frequency with which relapse ensues. In at least 25 per cent. of the patients who have recovered, either no relief has followed, or relapse has occurred subsequently. This happens especially when there is any active ulceration at the time of operation, while, even if there be no active ulceration, recurrence is not improbable for two reasons. First, gastroplasty is not a preventive of gastric ulcer, and does not modify the conditions predisposing to gastric ulceration, which presumably are present in these cases. Secondly, adhesions frequently are present which impair the motility of the stomach and keep up a certain degree of gastric stasis. This leads to hyperchlorhydria, which in its turn causes, or at least predisposes to, and keeps up, ulceration.

The same criticism applies to the operation of **gastro-gastrostomy**. At least 30 per cent. of the patients on whom this operation has been performed, either have obtained no relief, or have relapsed. It appears, therefore advisable,

that if gastroplasty or gastro-gastrostomy be the procedure adopted, gastro-jejunostomy should be performed in addition.

In my earlier cases I performed the double operation of gastroplasty and gastro-jejunostomy, but I think that in most cases this is unnecessary, and I now perform a gastro-jejunostomy in the proximal pouch. If pyloric stenosis exist as well as hour-glass stomach, my practice is to perform a double anastomosis by making a connection between the jejunum and both the cardiac and the pyloric pouches, as was suggested by Weir and Foote.

A simple gastro-jejunostomy, or in cases associated with pyloric stenosis, a double gastro-jejunostomy, has the merit that it does not necessitate cutting into, or operating on, the diseased area. In principle it seems the best method, and I consider it to be the method of choice. In cases of malignant hour-glass stomach, if practicable, partial gastrectomy should be performed.

Prognosis.—The prognosis after operation for hour-glass stomach is good, provided a gastro-jejunostomy be performed.

Owing to the extreme emaciation of many of those who are suffering from hour-glass stomach, the mortality rate is perhaps slightly greater than in gastro-jejunostomy for simple ulcer or for pyloric stenosis. Operation for hour-glass stomach should be a rare operation, as the patients should be treated surgically long before the ulcer has progressed to the extent of producing marked constriction of the stomach.

I have operated on fourteen patients suffering from hour-glass stomach, and so far have been fortunate in that all of them have recovered.

My first operation was performed in 1902, and all the patients except one have remained quite well ever since. The one exception remained quite well for five years, and then had symptoms of intestinal obstruction. I reopened

the abdomen and found that both the jejunum and transverse colon had been obstructed partially, at the site where they crossed one another. I performed an entero-anastomosis and an ileo-sigmoidostomy, and since then (nearly three years ago) the patient has remained in perfect health. In all my cases a gastro-jejunostomy was performed, in some of them combined with gastroplasty.

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CHAPTER XV

THE COMPLICATIONS OF GASTRIC AND DUODENAL ULCERS—*continued*

5. GASTRIC TETANY

Gastric tetany due to dilatation of the stomach was first recorded by Kussmaul, so recently as the year 1869.

In the majority of cases the dilatation has been due to cicatricial pyloric stenosis. In a few instances, the disease was in the first part of the duodenum (Reuvers, Baumberger, Dujardin-Beaumetz), in others, both the pylorus and the duodenum have been affected (Loeb, Müller).

In rare instances the obstruction of the pylorus has been due to malignant disease.

Often the early symptoms, pricking and numbness of the hands, pass almost unnoticed by the patient. Generally the first noticeable symptom is a tonic spasm of the muscles of the extremities. As a rule its onset is quite sudden, and is preceded by an attack of severe vomiting.

In 1902 I saw a typical case under the care of Dr. Soltau Fenwick. I will quote from my notes the description I wrote of the patient's condition during an attack : " Arms abducted, forearms flexed and strongly pronated. The wrists strongly flexed, the fingers drawn together, flexed at the metacarpo-phalangeal joint, the phalangeal joints extended. Thumb strongly adducted into the palm of the hand. Lower limbs fully extended, the ankles extended, and the soles of the feet adducted and rotated inwards. Some twitching about the mouth. Patient absolutely rigid all over, and unconscious."

The attack lasted six hours, during which time the patient remained unconscious, while the temperature gradually rose to 102·8° F., the highest point being reached just as the attack was passing off.

A few days after the patient had recovered from the attack I performed an anterior gastro-jejunostomy. The patient has never had a day's illness since the operation ten years ago, and he has put on three stone in weight. The case is of interest as it is, I believe, the first in England in which gastro-jejunostomy was suggested and performed for gastric tetany. In Scotland, a few months earlier, Mr. Caird had performed gastro-jejunostomy in a similar case, but it was not reported until a later date, so that when Dr. Fenwick suggested gastro-jejunostomy in this case, he was unaware that this operation had been performed previously for gastric tetany. Before this, several cases had been treated surgically, but in all of them pyloroplasty had been performed.

Usually certain symptoms are present. These are :

(i) **Trousseau's Symptom.**—As the attack is passing off, pressure over the principal nerve-trunk or blood-vessels, sufficient to impede the venous or arterial circulation, will cause a recrudescence of the paroxysm.

(ii) **Chvostek's Symptom.**—A slight tap over the principal nerve of the part affected will cause spastic contraction of the muscles supplied by the nerve.

(iii) **Erb's Symptom.**—Great increase of electrical irritability to a faradic current.

(iv) **Hoffman's Symptom.**—Slight pressure over a sensory nerve may cause paræsthesia in the area supplied by the nerve.

As the attack passes off there is considerable pain and tingling in the limbs. Usually the first attack is followed within a short time by others. Occasionally an interval of several days, or even months, separates the first and second attacks.

Slight albuminuria is an almost constant symptom, although no evidence of renal mischief has been discovered. The albuminuria is due, probably, to the excretion of some toxin, and is in favour of the view that the spasms are due to auto-intoxication, the result of abnormal chemical processes in the stomach (Bouveret and Devic). Marked tetany is a very serious complication of gastric ulcer, for about 70 per cent. of the cases recorded have ended fatally. At the same time it is a complication which ought not to occur. Prevention is better than cure, and the proper method of treating gastric tetany, is by dealing with all intractable gastric ulcers surgically. Fortunately, gastric tetany rarely is fatal during the first attack, but Marten has reported a case which proved fatal four hours after the first seizure, and Trevelyan has recorded a similar case. The dread of a second attack weighs on the patient's mind, and has a most depressing mental effect.

The treatment of gastric tetany, due to pyloric stenosis, is gastro-jejunostomy. The prognosis is good. Most of the patients have recovered, and have been relieved completely of their symptoms.

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CHAPTER XVI

THE PATHOLOGY, SYMPTOMS, AND DIAGNOSIS OF GASTRIC CARCINOMA AND SARCOMA

THE PATHOLOGY OF GASTRIC CARCINOMA

Frequency.—The stomach enjoys the unenviable distinction of being affected more frequently with carcinoma than any other organ of the body. According to Häberlin, 41·5 per cent. of 27,511 cases of carcinoma that ended fatally were carcinoma of the stomach. D'Espere found that 44·3 per cent. of all cancers are situated in the stomach. According to the Report of the Registrar-General, during the years 1901 to 1907 there were 207,764 deaths from malignant disease in England and Wales, and 16·8 per cent. of these deaths were the result of cancer of the stomach. Probably a considerable number of the deaths from gastric carcinoma are not certified as such.

During fifteen years, out of 39,568 patients admitted into St. Bartholomew's Hospital, in 183 the diagnosis was cancer of the stomach. In other words, of every 216 patients admitted, one was suffering from gastric carcinoma, or just under 0·5 per cent., a proportion almost identical with that found by other observers. Of these patients 118 were men and 65 women. Dr. Soltau Fenwick gives the proportion as rather less than six men to four women.

Gastric carcinoma is a disease of late middle life. It is rare in young people. Of the 183 patients referred to above, 58·8 per cent. were between the ages of forty and sixty years. According to the Registrar-General's

PLATE XLIII.
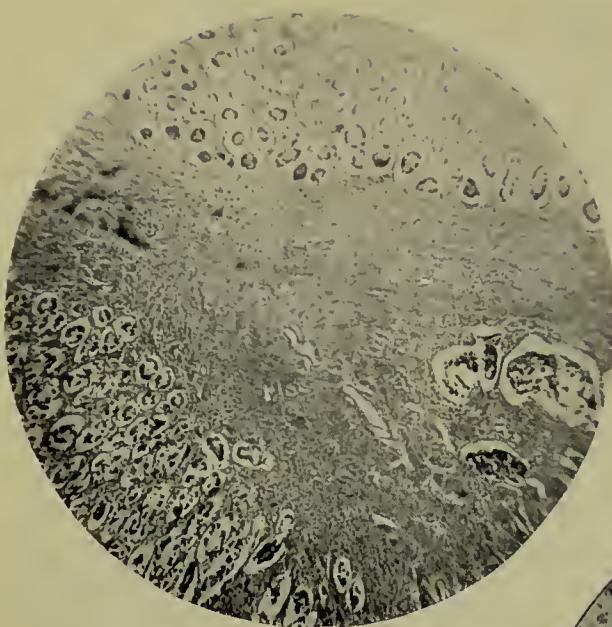
FIG. 62.—Microphotograph of
spheroidal-celled carcinoma.

FIG. 63.—Microphotograph of
a cylindrical-celled carci-
noma. (Soltau Fenwick.)
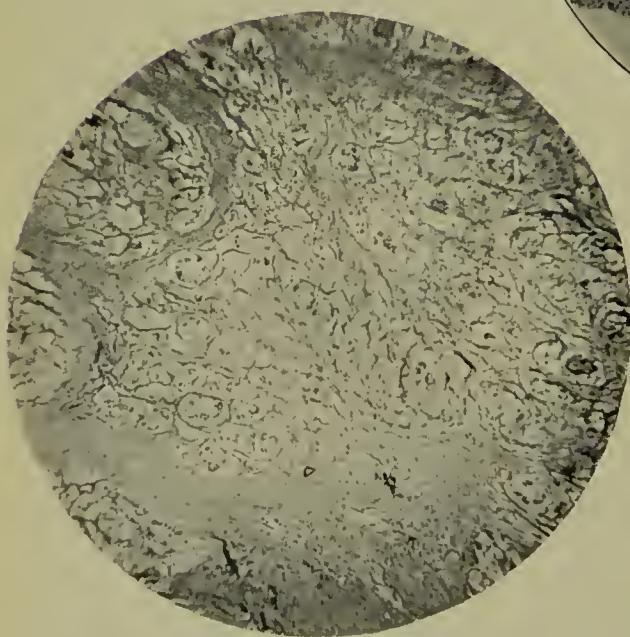
FIG. 64.—Microphotograph of carcinoma, showing
colloid degeneration.

returns, 79 per cent. of the patients who died from malignant disease of the stomach during the years 1901–1907, were between the ages of forty-five and seventy-five years.

Pathological Anatomy.—Two types of carcinoma occur in the stomach, the spheroidal-celled, and the cylindrical-celled or adeno-carcimona. In both types colloid degeneration may occur (Plate XLIII, Figs. 62, 63, 64).

Various estimates have been given as to the relative frequency of these two types. According to Cornil and Ranvier, cylindrical-celled is the more frequent. Dr. Fenwick concludes that the spheroidal-celled variety is more than twice as common as the cylindrical-celled. I have investigated the records of nearly two hundred cases in which an operation was performed, and an analysis of the records suggests that the cylindrical-celled variety is the more frequent. These cases, however, were not consecutive, and so cannot be accepted as conclusive evidence, but they do suggest that the cylindrical-celled type of carcinoma is more often amenable to radical treatment than is the spheroidal-celled.

At first the growth is limited to the gastric mucosa, but gradually it spreads to the submucous coat, and later extends to the peritoneum. The mucous surface of the growth ulcerates, and there is slight oozing of blood from the surface. In some cases the growth may destroy a large vessel, and so lead to alarming hæmorrhage.

The situation of the growth is important, not only from the point of view of surgical intervention, but because the symptoms depend, to some extent, on the part of the stomach affected.

The pylorus is more often implicated than any other part of the viscus. In the following table is shown the situation of the growth according to various authorities, together

with the analysis I have made from 168 consecutive post-mortem examinations at St. Bartholomew's Hospital.

TABLE SHOWING THE POSITION OF GROWTH AS CALCULATED
BY WELCH, BRINTON, HABERSHON, FENWICK, AND THE
AUTHOR.

*In order to compare their statistics with mine I have expressed
all the tables in percentages.*

	Welch.	Brinton.	Habershon.	Fenwick.	Author.
Pylorus	60·8	60·8	51·9	58	61·3
Lesser curvature . . .	11·4	10·5	13·9	11·5	14·2
Cardiac orifice . . .	8·0	10·0	12·6	9·8	5·9
Anterior wall . . .	2·3	3·0	6·3	2·2	5·3
Whole stomach . . .	4·7	3·6	5·0	6·0	4·7
Central	...	1·1	5·0	...	4·1
Posterior surface . . .	5·2	3·0	...	5·0	4·1
Greater curvature . . .	2·6	3·0	·2	2·8	...
Not stated	...	4·7	1·2	...	...

It is clear, then, that the growth far more frequently affects the pylorus and adjacent portion of the stomach than any other, the lesser curvature being the next most common seat of new growth.

When the growth is situated at the pylorus, it leads, sooner or later, to obstruction, which in some cases may be almost complete (Plate XLIV, Fig. 65).

In a few cases, the growth, instead of being limited to the pylorus and its immediate neighbourhood, infiltrates the wall of the stomach in its whole extent (Plate XLIV, Fig. 66). In these cases the stomach is sometimes small and densely hard, and from its shape is called " leather-bottle stomach."

When the peritoneum is invaded, the growth becomes adherent to neighbouring structures (liver, pancreas, colon, anterior abdominal wall), and when the whole thickness of the gastric wall is destroyed by ulceration, the base of the malignant ulcer is formed by the organ to which it has become adherent. When this is one of the hollow viscera,

PLATE XLIV.
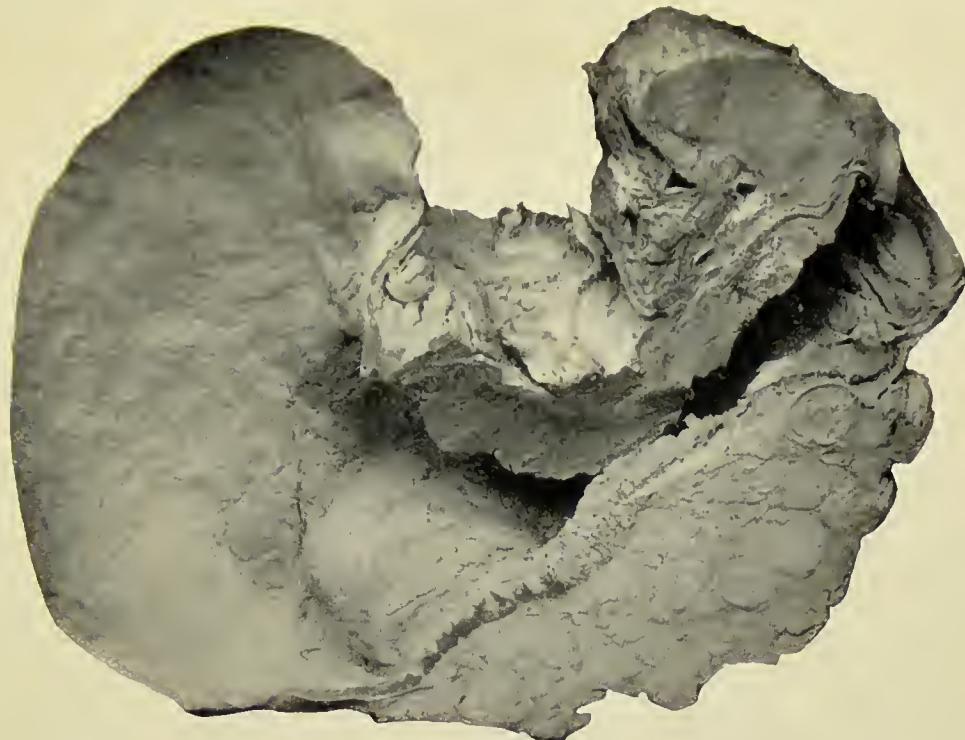
FIG. 65.—Cylindrical-celled carcinoma of the pylorus which has resulted in stenosis. Viewed from behind. (Soltau Fenwick.)
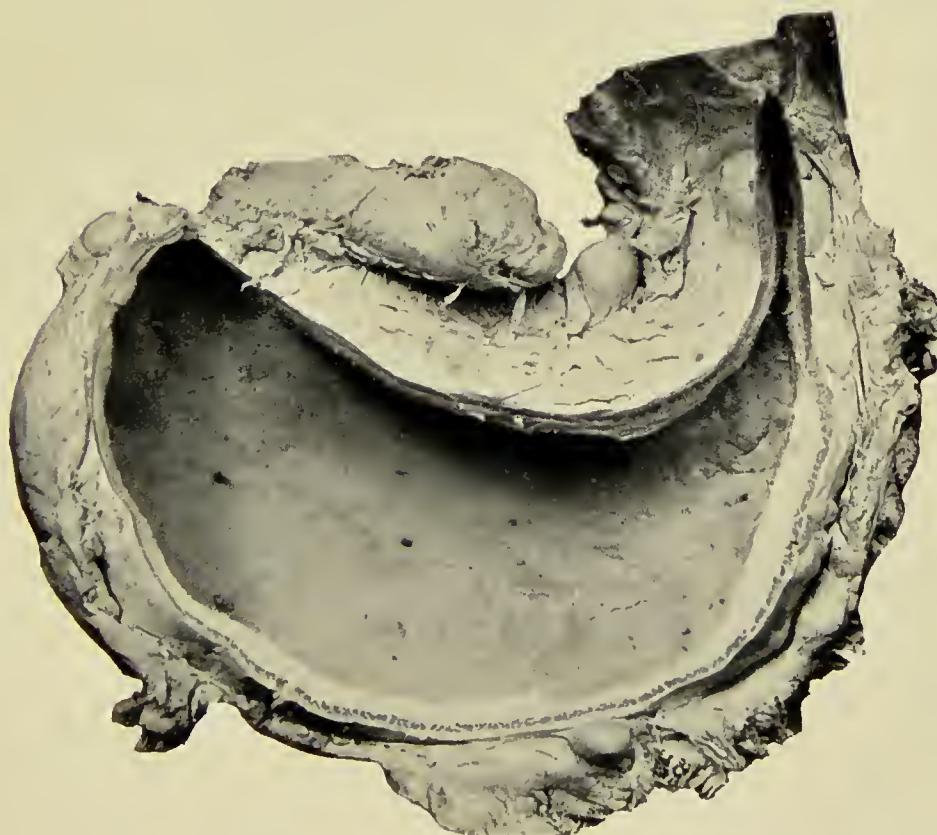
FIG. 66.—A stomach viewed from behind, showing diffuse infiltration by scirrhus carcinoma, with enlarged glands along the curvatures. (Royal College of Surgeons' Museum. No. 2408a.)

a fistula may be formed between the stomach and the viscus to which it has become adherent (*e.g.* gastro-colic fistula).

The Lymphatic Glands in Gastric Cancer.—The stomach is supplied richly with lymphatic vessels. According to Cuneo the lymphatic spaces between the gastric glands are intercommunicating, form large spaces in the mucosa, pass
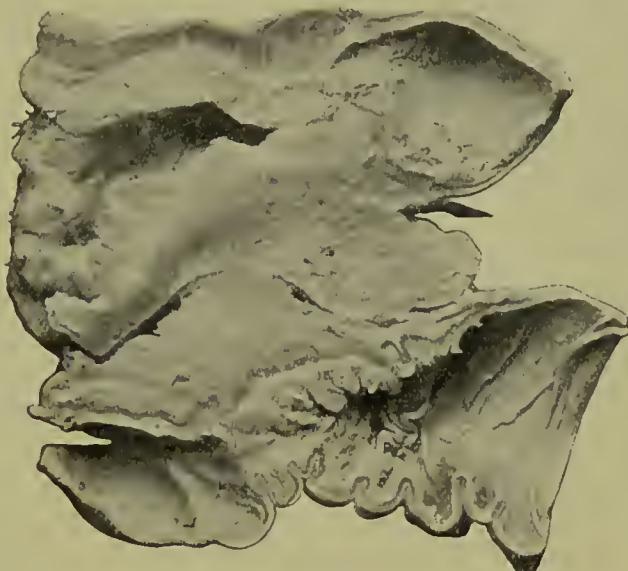
FIG. 67.—Colloid carcinoma of the pylorus invading the transverse colon. Specimen viewed from behind.   (Soltau Fenwick.)

through the muscular coats, collect in the subserous layer, and enter lymphatic channels just under the peritoneum.

The lymphatic vessels of the stomach form three principal groups.

1. Those passing upwards towards the lesser curvature, which drain the upper half of the pyloric two-thirds of the stomach.

2. Those passing downwards towards the greater curvature, which drain the lower half of the pyloric two-thirds of the stomach.

3. Those passing across the fundus, which they drain, enter the gastro-splenic omentum and terminate in the glands of the hilum of the spleen.

The glands into which the lymphatic vessels from the stomach drain, may be divided into four groups (see Fig. 68).

(*a*) The glands along the lesser curvature (**gastro-hepatic group**), situated in the gastro-hepatic omentum. Their efferent vessels pass backwards near the cardiac orifice and enter the glands around the cœliac axis.

Fig. 68.—Diagram to show lymphatic glands of stomach.
A. Glands of lesser curvature (gastro-hepatic glands).
B. Sub-pyloric glands.
C. Glands of greater curvature (gastro-colic glands).

(*b*) The glands along the lower border of the pylorus (**sub-pyloric group**). Their efferent vessels pass into the cœliac glands.

(*c*) The glands behind the pylorus, around and over the head of the pancreas (**retro-pyloric group**). Their efferent vessels pass to the cœliac glands.

(*d*) The glands along the greater curvature (**gastro-colic group**), situated in the gastro-colic omentum along the lower border of the stomach. Their efferent vessels pass into the glands around the cœliac axis.

PLATE XLV.

FIG. 69.—The stomach and transverse colon, showing the areas drained
by the different groups of lymphatic glands. The arrows show the
direction of the lymph current. (W. J. Mayo.)
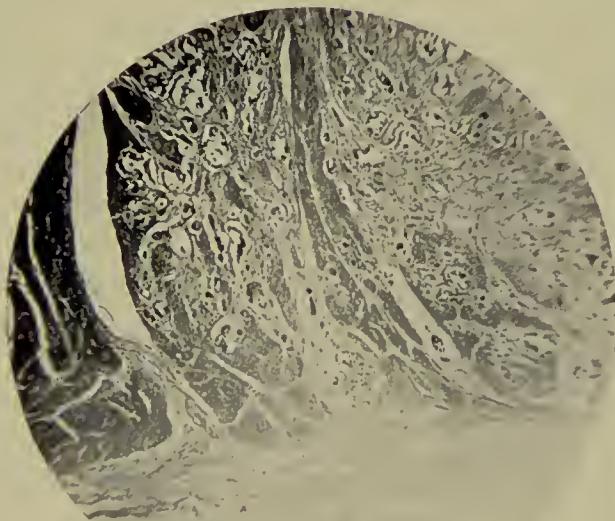
FIG. 70.—Microphotograph of a section cut beyond the area of induration
in a case of gastric carcinoma, showing islets of growth infiltrating
the muscular tissue.

A practical point, the importance of which will be indicated later, is, that the glands along the greater curvature communicate with the glands lying along the upper border of the transverse colon.

As carcinoma usually is situated close to the pylorus at the lesser curvature, the gastro-hepatic glands are the first to become enlarged ; sooner or later all the groups above-mentioned may become affected. The circumstance, that the glands are enlarged, does not necessarily mean that they are the seat of carcinomatous deposit; the enlargement may be due to inflammatory infiltration. Quite small glands may contain carcinomatous deposit, while glands of considerable size may be simply inflammatory, a very important point in considering the advisability of resection. In carcinoma of the cardiac orifice the infection of the lymphatic glands of the stomach is slight, but the deep coeliac glands may be implicated at an early stage of the disease. A very valuable paper has been published recently by Dr. MacCarty on the lymphatic glands in stomach cancer. His conclusions are as follows :

A. Negative Conclusions :

(i) The size of regional lymphatic glands bears no apparent relation to the size of the primary lesion in the stomach.

(ii) The size of a lymphatic gland is no criterion of the presence or absence of carcinoma.

(iii) Gross diagnoses of lymphatic glands are of no value except in advanced carcinoma of the glands.

(iv) The duration of symptoms bears no apparent relation to the size and extent of involvement in the lymphatic glands.

(v) The average age at operation and sex bear no direct relation to the glandular involvement.

B. Positive Conclusions :

(i) The average loss of weight increases with the increase in extent of glandular involvement.

(ii) The immediate hospital post-operative mortality is in direct proportion to the amount of glandular involvement.

(iii) The subsequent mortality is in direct proportion to the amount of glandular involvement.

(iv) Carcinomatous glandular involvement is very often microscopic.

(v) The surgeon who desires to treat early carcinoma must depend upon the microscope in the hands of an experienced pathologist for early carcinomatous lymphatic involvement.

(vi) The diagnosis of early carcinomatous involvement requires extensive experience in the study of so-called pre-carcinomatous reaction of lymphatic glands.

The Spread of Gastric Carcinoma.—From a surgical point of view, knowledge of the manner and direction in which the disease spreads, is of considerable importance.

Gastric carcinoma spreads in three ways :

(i) **By Direct Continuity.**

(ii) **By Contiguity, or Invasion of Surrounding Structures.**

(iii) **By Dissemination through the Lymphatics.**

(i) **By Direct Continuity.**—It is a curious fact, to which attention was directed first by Brinton and Rokitansky, that cancer of the stomach rarely spreads far into the duodenum. This is in striking contrast to the course of carcinoma of the cardiac orifice, as in this situation, the growth almost invariably spreads directly into the œsophagus.

Another practical point in connection with extension by continuity is, as shown by Czerny and Cuneo, that islets of cancer-cells may be found in the submucous coat *beyond* the area of infiltration. This is seen well in the microphotograph (Plate XLV, Fig. 70), which is taken from a section half an inch beyond the indurated area. A group of cancer-cells is seen infiltrating the muscle.

In order to be certain of removing the disease completely,

the section should be made at least two inches beyond the indurated area.

The disease spreads along the lines of the lymph current, and as the greater number of lymphatic vessels run towards the lesser curvature, it follows that the growth extends in this direction. Thus the lymphatic glands along the lesser curvature may be affected by direct continuity. Consequently, it is of little use to dissect them out; they must be removed in one mass, together with the portion of stomach wall to which they are attached. If this be not done, the tributary vessels will be cut across, allowing the escape of cancer-cells which may infect the tissues with which they come in contact, and so lead to an early recurrence.

The growth may also spread by direct continuity along adhesions which have resulted from localised inflammation of septic origin. These facts emphasize the importance of wide removal of the disease whenever excision is performed.

The lymphatic vessels and glands of the dome of the stomach rarely are infiltrated. This portion of the stomach, for which Sir Berkeley Moynihan has suggested the term " isolated area," usually may be left with safety.

(ii) **By Contiguity.**—Spread of the disease by contiguity may occur in two ways (*a*) by implantation on a contiguous surface, or (*b*) by invasion of a neighbouring structure along adhesions.

(iii) **By Dissemination.**—Dissemination occurs through the lymphatic vessels. The liver is the organ affected most frequently (36 per cent. of the cases in my series).

The cases most suitable for radical treatment are those in which the growth is limited to the pyloric end of the stomach.

When the growth has invaded the duodenum, or has spread by continuity to the gastro-hepatic and gastro-colic glands, complete removal of the disease is practicable in

Q

many instances. When the growth has invaded surrounding structures by contiguity, *e.g.* the pancreas, complete removal is very difficult, often impracticable. When the growth has become disseminated to other organs, radical cure is impossible. Local removal can be only a palliative measure.

Unfortunately, in a large number of instances, when the patient first is seen by the surgeon, the growth is too advanced for radical cure.

It is a lamentable truth that many patients are allowed to die, who might have been relieved, if not cured, by a timely excision. From investigations I have made, I believe that in 20 per cent. of the fatal cases, there is no extension of the disease by contiguity, and no secondary deposits are discoverable at the post-mortem examination.

THE SYMPTOMS OF GASTRIC CARCINOMA

There is no sign or symptom pathognomonic of cancer of the stomach. In the early stages the symptoms are few and slight; in the later stages so plain, that he who runs may read.

Many of the so-called symptoms, such as ascites, jaundice, thrombosis, are late complications. When such are present, the opportunity for successful surgical treatment has gone for ever. To be of real benefit to the patient, the diagnosis must be made before these complications are present.

A discussion on these later so-called symptoms would serve no useful purpose. I propose, therefore, to deal with the symptoms of the earlier stages only, in which radical treatment may be possible.

The earliest symptoms of cancer of the stomach are: pain or discomfort after food, loss of appetite, failure of strength, anæmia, and loss of weight.

Pain, or rather a feeling of discomfort and uneasiness after food, usually is the first symptom which attracts the patient's attention, insidious and scarcely perceptible at first. In some cases the onset is comparatively sudden, perhaps following some indiscretion in diet. At first its onset is determined by food, but as it increases in severity, it becomes more or less constant. It is accompanied by flatulence, and belching of gas. The pain is described as of a dull, sickening character, is referred to the epigastrium, and, as a rule, is less severe than the pain of gastric ulcer. Radiation of the pain is a late symptom, and means that the growth is invading the liver or pancreas. According to Dr. Fenwick, in 14 per cent. of the cases pain is either absent or extremely slight during the whole course of the malady.

Loss of appetite is gradual in its onset. The patient refuses food, not because it causes him pain, but because his appetite is gone. In many instances the patient evinces a marked dislike for meat.

Progressive loss of strength is an early symptom, sometimes the first to attract attention, and often is associated with impaired mental activity, and depression of spirits.

As a rule, even in the early stages of the disease, a slight but increasing degree of **anæmia** is present. In some cases a marked degree of anæmia is the earliest symptom of the disease. Such cases, in my experience, run a very rapid course, and are beyond the reach of surgery before a diagnosis is made. Fortunately, these form only a small proportion of the total number.

Even in the early stages of gastric carcinoma, almost invariably there is a gradual and progressive **loss of weight.** If, however, the patient be kept in bed, and be dieted carefully, in some instances the body weight may be maintained or even increased. This is a very important point, for I am convinced that a good many cases of cancer of the stomach

are overlooked, because with rest in bed and dieting, the patient improves temporarily and puts on weight. It is not uncommon in hospital notes to find a record such as this: " The patient was admitted for supposed cancer of the stomach, but under medical treatment gained in weight, and was discharged much improved." Later, the patient comes under observation again, with obvious and inoperable gastric cancer.

I have observed a patient with cancer of the stomach put on as much as four pounds in weight in ten days. Such improvement is likely only in early cases, before the disease has spread beyond the limits of the pylorus—in other words, just those cases which would be benefited most by operation.

I wish, therefore, to lay particular emphasis on this point—namely, that gain in weight and temporary improvement under medical treatment and rest in bed, does not contra-indicate a diagnosis of cancer of the stomach.

The Presence of Blood in the Fæces.—The persistent presence in the fæces of blood, so small in amount that it is not recognisable macroscopically (occult blood), is very suggestive of gastric carcinoma. When the mucosa over a carcinomatous growth has become ulcerated, usually there is slight but persistent hæmorrhage, which can be recognised in the fæces by special tests. Ulcer may lead to the presence of occult blood, but in this disease the hæmorrhage is irregular and intermittent. It is only if the presence of blood be constant and persistent, that it is significant of cancer. For the test to be of value, the fæces must be examined after the patient has been on a blood-free diet for at least three days. The method of carrying out the test is described in the Appendix (p. 331).

A Palpable Tumour is not an early sign of cancer. In a few cases, however, the detection of a tumour is the first evidence of the disease, the other symptoms having passed

PLATE XLVI.

FIG. 71.—Carcinoma of the stomach, examined on the couch
after bismuth, showing irregularity due to the growth. *C., P.,*
cardiac and pyloric portions of the stomach; *a, b, c, d,* the four
parts of the duodenum; *U,* umbilicus marked by a penny.—By
Dr. A. C. JORDAN.

FIG. 72.—Carcinoma of the stomach. The lumen of the stomach is
encroached upon by a nodular growth in the wall of the viscus,
with islands or projections into the normal tissue giving the
appearance of finger prints. (By Dr. Lewis Gregory Cole.)

unnoticed. This possibility emphasizes the importance of a careful and systematic examination of the abdomen in all cases of gastric trouble.

In palpating for a tumour the abdomen should be examined, not only after emptying the stomach, but after distending it with air. In some cases in which this is done, a hardening can be felt in the region of the pylorus, probably due to a spasm of the pylorus set up by the distension. When present this sign is an indication, not necessarily of cancer, but of the necessity for surgical treatment.

Our aim should be to establish a diagnosis, or at least to decide on the necessity of surgical treatment, before a tumour can be felt. Nevertheless a palpable tumour by no means contra-indicates the possibility of complete removal. One of my patients, who had a large tumour, not only palpable but visible, lived nearly four years after partial gastrectomy.

Hæmorrhage, as evidenced by hæmatemesis or by melæna, is a late, rather than an early symptom of gastric cancer.

Vomiting is an occasional symptom in the early stage of carcinoma of the stomach, but when the growth has produced pyloric stenosis, it is a constant and distressing feature. Usually the vomit is dark brown in colour, very acrid or offensive to the smell, and may contain blood in considerable quantity.

Although the existence of pyloric stenosis premises that the disease has progressed beyond the early stage, its presence does not negative the possibility of resection, because, when stenosis is marked, the condition of the patient is such, that a diagnosis of the necessity for surgical treatment is made at an earlier stage than when there is no obstruction at the pylorus.

In Carcinoma of the Cardiac End of the Stomach, the early symptoms are very indefinite. Usually, difficulty in

deglutition is one of the earliest signs. The patient notices that he cannot swallow solid substances with ease, or as it is expressed by the patient, "the food sticks on its way into the stomach."

Examination of the Gastric Contents

When interpreted with the clinical history of the case, gastric analysis is of the greatest help, often the chief aid in assisting us to come to a conclusion as to the necessity for surgical treatment.

The three most important points are :

1. **Evidence of Impairment of the Motor Functions of the Stomach.**

2. **Alteration of the Amount of Chlorides in the Gastric Juice.**

3. **Impaired Digestion of Albumen.**

1. Evidence of Impairment of the Motor Functions of the Stomach

In 65 per cent. of the cases of gastric carcinoma, the disease begins at, or near, the pylorus. It follows, therefore, that one of the earliest signs of cancer of the stomach, is impairment of its motility. It is rare, even in the earliest cases, to find the stomach empty in the early morning. On passing a stomach-tube a varying amount of darkish fluid is withdrawn. Sometimes the quantity withdrawn amounts to 200 c.c. or 300 c.c., and has a very offensive smell. In the early morning contents of the stomach, free hydrochloric acid occasionally is present, in contrast to the usual absence of free hydrochloric acid in the gastric contents after a test-meal.

The quantity, moreover, of the gastric contents, withdrawn one hour after the test-meal has been given, usually is considerably above the average ; the toast is ill-digested, the fluid contains a large amount of mucus, and consequently

is so thick and ropy that the gastric contents are difficult to pour. Often it is possible to diagnose malignancy simply by inspection of the contents drawn off after a test-meal.

As a rule there is a considerable increase in the amount of the volatile acids, which vary from 5 per cent. to 20 per cent.

2. The Alteration in the Amount of Chlorides

(*a*) **Diminution of the Total Chlorides.**—The amount of total chlorides found by analysis after a test-meal, invariably is less, and usually considerably less, than normal. In my experience the amount is never more than 0·300, and in about half the cases is less than 0·200.

(*b*) **Absence of Free Hydrochloric Acid.**—As a rule free hydrochloric is absent from the stomach contents in gastric carcinoma. For long this sign was regarded as pathognomonic of cancer. Now we know that this is not the case. Free hydrochloric acid often is absent in cases which are not malignant. Nevertheless, absence of free hydrochloric acid is the rule, presence of free hydrochloric acid the rare exception. I believe that when free hydrochloric acid is present, the growth is more likely to be situated at the cardiac end of the stomach.

(*c*) **Diminution of the Protein Hydrochloric Acid.**—A marked diminution of the protein hydrochloric acid, in my experience, is a very early symptom of gastric cancer. It is true that in chronic gastritis, due to causes other than malignant disease, there is marked diminution of the protein hydrochloric acid, but in estimating the value of this sign, we must consider its importance in relation to the clinical history. Chronic gastritis, secondary to cancer, progresses much more rapidly than gastritis due to other causes.

The relation of the degree of diminution of the protein hydrochloric acid to the duration of the patient's symptoms,

must be considered. In other words, marked impairment of gastric motility, or marked diminution of protein hydrochloric acid, in a patient over middle age, raises a suspicion of malignancy, and the shorter the history and the greater the degree of gastritis, as shown by analysis, the greater the probability of cancer. Let me illustrate this by giving the analysis of the gastric contents of two patients, neither of whom had any palpable tumour.

No. 1

Total acidity	44
Total chlorides	0·206
Free HCl	0·000
Protein HCl	0·082
Mineral chlorides	0·124

The above analysis was made from the gastric contents of a man who had suffered from gastric symptoms for a year. The chemical findings are those of a chronic gastritis, with very marked diminution of the protein hydrochloric acid. This might be due to inflammatory diseases or cancer. The comparatively long history, and at the same time the fair condition of the patient, and the absence of any degree of anæmia, were against malignant disease. Cancer of such standing should have resulted in a much lower total acidity, and in a more marked diminution of the protein hydrochloric acid. At the operation a gumma of the liver was found which had become adherent to, and implicated the pylorus.

No. 2

Total acidity	29
Total chlorides	0·183
Free HCl	0·000
Protein HCl	0·070
Mineral chlorides	0·113

This analysis was from the gastric contents of a woman who had suffered for four months from symptoms referable to the stomach.

In this case the degree of gastritis was much greater than in the first case, and yet the history was much shorter. This, coupled with the fact that the patient evidently had lost flesh, and was slightly anæmic, led to a diagnosis of cancer, which was confirmed by operation, and the growth successfully removed.

I have not, as yet, met with a case in which the protein hydrochloric acid was about 0·100 or less, in which there was not some condition requiring surgical treatment.

(d) **Increase of Mineral Chlorides.**—The mineral chlorides as a rule, are increased, the amount being generally more than 0·100. Seeing that the amount of the total chlorides is diminished, it is probable that the increase of the mineral chlorides is due to neutralisation of free hydrochloric acid by an alkaline fluid secreted by the growth.

Usually the total acidity of the gastric contents is low, varying from fifteen to forty.

3. Impaired Digestion of Albumen

In gastric carcinoma the digestion of albumen, as tested by the carmine method, or by Hammerschlag's method, invariably is impaired. This impairment is not pathognomonic of carcinoma, but taken in conjunction with the history and other clinical signs, is a valuable adjunct to our means of diagnosis.

Other important points which can be ascertained by examination of the gastric contents are :

The Presence of Unusual Substances in the Gastric Contents

(a) **Lactic Acid.**—For lactic acid to be present in the stomach in excessive amount, four conditions are necessary, namely, gastric stagnation, absence or great diminution of

the secretion of hydrochloric acid, impaired absorption, and reduced albumen-digesting power.

The presence of excess of lactic acid is not pathognomonic, but it is a valuable confirmatory sign, present, according to Dr. Fenwick, in 91 per cent. of all cases of cancer of the stomach. In my experience, it is not present in excess in early cases. The sign is of value only if the acid be present after a Boas' test-meal (see p. 10).

(*b*) **The Oppler-Boas Bacillus.**—Both Riegel and Hemmeter regard the presence of the long, non-motile Oppler-Boas bacillus of considerable importance. Hemmeter and Adler found the organism present in 52 out of 55 cases of gastric carcinoma examined, while in 18 cases of gastric ulcer it was absent. According to Kauffman and Schlesinger the presence of a large number of bacilli is an indication of carcinoma.

(*c*) **The Bacillus Filiformis.**—According to Dr. Ehret the existence of cancer can be affirmed when masses of this long, slender, thread-like bacillus are found in the contents of the fasting stomach, provided that the contents of the fasting stomach are only moderate in quantity.

(*d*) **Fragments of Growth.**—The presence of fragments of growth in the gastric contents necessarily is diagnostic of cancer, but the presence of such fragments implies that the disease is advanced, and so is not of much value for the purpose of early diagnosis.

(*e*) **Cancer Cells.**—Hemmeter attaches considerable importance to the examination for cancer cells; he feeds the patient per rectum for forty-eight hours, and then washes out the stomach with saline solution. He uses a stomach-tube with a sharp eye which scrapes away minute portions of the gastric mucosa. The washings are centrifugalised and then examined microscopically. Cells in a state of atypical mitosis may be found. Personally, I must

confess that I have not found any of these last four signs of much value in the early diagnosis of gastric carcinoma.

The Examination of the Urine in Gastric Carcinoma

1. **Excretion of Uric Acid.**—It is possible that the examination of the uric acid excretion may prove of value in the diagnosis of gastric carcinoma. In a healthy individual on a milk diet the amount of uric acid excreted varies from 0·1 to 0·3 grammes. In carcinoma, even on a milk diet, there is a considerable increase in the amount excreted; as much as 0·8 or even 1·0 gramme may be found in the urine collected during the twenty-four hours. So far as my observation goes, this is not so in gastric ulcer or in chronic gastritis, but whether this increase obtains in other conditions, as yet, I have not been able to determine.

2. **Decrease in Phosphates.**—Miss Elsie M. Royle has done some suggestive work in connection with urine in malignant disease. Miss Royle concludes as follows :

" 1. The urine of a patient suffering from cancer is usually richer in uric acid than the urine of healthy persons under similar conditions.

" 2. The output of phosphates in the urine is, in the majority of cases of carcinoma, less than is the case with healthy individuals.

" 3. The ratio of phosphates to uric acid is almost invariably reduced in malignant disease below that found in health, any ratio below four being suggestive, especially if it remains low on several occasions. Any ratio below three is almost diagnostic of malignant disease, or of a blood disease—unless, indeed, further work proves that the same holds good for other conditions."

THE DIAGNOSIS OF GASTRIC CARCINOMA

It is, I believe, to chemical pathology that we must look for the solution of the problem—How to diagnose gastric carcinoma in its early stage. At present examination of the gastric contents is our chief help, but it is not conclusive.

Dr. John B. Deaver, in urging earlier diagnosis of cancer of the stomach, says that the greatest source of delay is the test-meal, and that prolonged and useless laboratory investigation is responsible for the hopeless condition in which so many of the patients present themselves to the surgeon. I do not think that this is a fair way of stating the case. There is no need for *prolonged* investigation. No examination which is likely to prove of value need occupy more than a few days. It is the delay in giving the test-meal, not the delay due to the giving of the test-meal, which is responsible for so many inoperable cases. I am convinced that with routine use of the stomach-tube for diagnostic purposes in all cases of persisting indigestion, the number of inoperable cases of gastric cancer would be diminished greatly, the operation mortality reduced, and the number of cures proportionately increased.

Apart from finding fragments of the growth, or definite cancer cells in the stomach washings, our only certain means of diagnosis lies in abdominal exploration. The practical question then is, Under what circumstances is this justifiable ? Are we to open the abdomen on suspicion, on probability, or only on certainty of the diagnosis ? If we wait for certainty, in most cases we have allowed the opportunity for radical treatment to pass.

Are we to Open the Abdomen on Suspicion ?—There must be suspicion of gastric carcinoma when a patient past middle life complains of persistent stomach trouble, with an onset, more or less abrupt, and without apparent cause. If, in spite of two or three weeks' treatment, improvement do not

occur; if we find progressive diminution of the gastric acidity and digestive power, or any other of the special signs to which I have alluded, suspicion becomes probability, and exploration should be delayed no longer. There is no doubt that many cases of gastric carcinoma are treated by medical means for long periods, in the hope that they may prove innocent. Such hope rarely is justified. In most of these cases, the condition found is such, that even if it be inflammatory and not malignant, relief can be obtained only by surgical means.

How many cases of pyloric stenosis, both innocent and malignant, are treated for many weeks by gastric lavage, and yet the futility of such treatment, as a curative measure, is well known! In an elderly person, the probability that even a slight degree of gastric stasis is due to carcinoma, is so great, that its persistence, in spite of careful dieting, renders exploration justifiable. **When once we are satisfied as to the probability of cancer, the risk of abdominal exploration is less than the risk of delay.** We must not shut our eyes to the axiom, that opening the abdomen is attended with risk, but the risk is reduced to a minimum, *if* we are content to limit our exploration.

If the disease be in a situation which permits of radical treatment, it is unlikely to be overlooked. The probability is, that a growth which cannot be palpated readily at an exploratory operation is inoperable, and its non-discovery of little practical importance. Some years ago I opened the abdomen of a man whom I supposed to be suffering from gastric carcinoma. I could find no growth. Two months later enlarged glands were noticed above the left clavicle. Some six months later he died from a carcinoma at the junction of the œsophagus with the stomach. Such cases are no argument against the value of abdominal exploration, but are an argument for tempering our zeal with discretion,

and limiting both the extent and duration of our exploration. No one is more averse than I am from performing an " exploratory operation." At the same time, I think that the really reckless surgeon is not he who explores, after considering carefully the clinical history, and the results of gastric analysis, but he who adopts the policy of " wait and see," until the possibility of performing a radical operation is gone for ever.

There are occasions on which the symptoms, while uncertain, yet are so suggestive that exploration should not be postponed.

The information gained by exploration is not always conclusive. Many cases are on record in which, at the operation, the disease was considered to be malignant, but has subsequently proved to be inflammatory. Fortunately, in such cases the situation and relations of the growth are such that partial gastrectomy, as a rule, is out of the question.

With increasing experience, I believe that in the great majority of cases it is possible to distinguish inflammation from malignant disease, but it must be admitted, that in a few, differentiation is impossible.

To sum up : exploration is indicated as soon as there is a probability of gastric carcinoma. Probability is established when a patient in the middle period of life, suffers from gastric trouble which is progressive, unrelieved by treatment, and accompanied by any two of the following signs :

1. Absence of free hydrochloric acid, and impaired digestive power.

2. Marked diminution of the protein hydrochloric acid.

3. Constant presence of blood in the fæces.

4. Gastric stasis and impaired motility.

5. Abnormal excretion of uric acid.

I have not said anything about the gastroscope as an aid to diagnosis, because, although it is an exceedingly ingenious and interesting instrument scientifically, I am doubtful

whether it will be of service in the diagnosis of cancer. Considering how difficult sometimes it is to decide whether a swelling be inflammatory or malignant, even when we can feel and see it, I do not think we can expect much help from an instrument which aids us by sight alone.

Differential Diagnosis

The following are the principal conditions which may give rise to difficulty in diagnosis : ulcer and its consequences, gall stones, severe anæmia, syphilis of the stomach, tubercle, and Bright's disease.

1. **Ulcer and its Consequences.**—When an extensive ulcer is present the symptoms may simulate closely those of cancer. There is emaciation, hæmorrhage, and consequent anæmia, absence of free hydrochloric acid, and impairment of the gastric motility. In the case of an ulcer, however, the percentage of the protein hydrochloric acid rarely is as low as in carcinoma. In cancer the loss of appetite is an early symptom, while the appetite of a patient with an ulcer remains good until a late stage, but he does not eat for fear of the pain caused by food. In the case of ulcer, the loss of flesh, and the failure of strength, are in proportion to the duration of the disease, and to the extent of abstinence from food. When cancer is present the loss of strength is more marked, progresses more rapidly, and is greater than can be accounted for by the diminished amount of food taken. When an ulcer of the duodenum has resulted in much contraction of the pylorus, or a gastric ulcer has produced an hour-glass stomach, the emaciation is so pronounced that a suspicion of malignancy is raised. The washing-out test (see p. 224), and the results of gastric analysis, usually will settle the diagnosis, although it must be remembered that occasionally malignant disease of the body of the stomach causes hour-glass contraction.

The most difficult cases of carcinoma to diagnose are those in which there is a history of stomach trouble extending over many years. In such, as a rule, careful cross-examination will elicit the fact that there has been an exacerbation of the symptoms, which lately have become continuous, so that the patient no longer experiences the temporary remissions which occurred in the past.

It is, no doubt, as I have said already, difficult to distinguish gastric carcinoma from inflammatory disease, but there is no difficulty in deciding in which cases surgical treatment is indicated. The clinician may be forgiven if he fails to diagnose cancer, but failure to recognise the necessity for surgical treatment is less excusable.

2. **Gall Stones.**—Very occasionally the symptoms in the late stages of gall-bladder disease resemble those of gastric cancer. If we can obtain an early history of sudden, severe, irregular attacks of epigastric pain having no relation to food, we may be able to eliminate cancer from our choice of diagnoses. Gastric analysis, too, is helpful, as, although exceptionally, free hydrochloric acid may be diminished or absent in cholelithiasis, there is little or no marked diminution of the protein hydrochloric acid, and the total acidity of the gastric contents is not as low as in carcinoma.

3. **Anæmia.**—Dyspeptic symptoms and loss of appetite may accompany anæmia, and suggest malignant disease, a suggestion which may be supported by the gastric findings. In anæmia there is pallor, and a waxy appearance of the skin, and often puffiness of the face. Examination of the blood reveals poikilocytosis, low hæmoglobin, and a high-colour index. The spleen may be enlarged. In cancer the percentage of hæmoglobin is either normal, or above normal, and the colour index usually low.

4. **Syphilis.**—Syphilis of the liver or stomach in the early

stages may simulate ulcer, and in the later stages, when cachexia is marked, may suggest cancer. On analysing the gastric contents the percentage of the protein hydrochloric acid often is found to be low, but the total acidity is higher than is the case in malignant disease. The patient's general condition, moreover, is much better than would be the case in gastric carcinoma of similar duration. The following case illustrates very well how the two conditions may be differentiated :

A man, aged thirty-three, under the care of my colleague, Dr. S. Fenwick, had suffered from pain, and a feeling of fullness after meals, for about a year. Some months before admission he began to suffer from vomiting, the vomit often being greenish in colour and containing altered food. The patient had lost flesh considerably. There was a history of syphilis. On admission there was a movable, insensitive swelling about the size of a walnut, above and to the right of the umbilicus. The swelling moved with respiration, and could be distinguished from the liver. The stomach was dilated considerably. The patient was very thin and sallow, but otherwise healthy and in good condition.

Gastric Analysis

Total acidity	44
Volatile acids	9·6
Total chlorides	0·206
Free HCl	0·000
Protein HCl	0·082
Mineral chlorides	0·124

The chemical findings were those of chronic gastritis, suggesting malignant disease.

The absence of marked anæmia, and the good condition of the patient, considering the long history, were against malignant disease. The diagnosis made was " inflammatory pyloric stenosis." At the operation I found a hard, nodular mass under the surface of the right lobe of the liver, extending backwards for some

distance. On incising this, some gummatous material escaped, suggesting a gumma. A portion frozen and microscoped at once showed no evidences of malignant disease. The cavity was drained. The duodenum was adherent to the under surface of the mass, and was obstructed by adhesions. Posterior gastro-jejunostomy was performed, and the patient was completely relieved, and has, since his operation five years ago, remained in perfect health.

5. **Tuberculosis.**—Tuberculous ulceration of the intestine may, in its later stages, suggest gastric carcinoma. This is the case especially when the ulceration results in stricture of the intestine. Emaciation, pain after food, loss of appetite, and vomiting, make a clinical picture closely resembling cancer. In addition the findings from analysis of the gastric contents are those of malignant disease, except that the motility of the stomach is not impaired to the same degree as in cancer. The slow onset of the disease, the hectic, irregular temperature, suggest tubercle rather than cancer, and by the time that the disease is sufficiently advanced to resemble cancer, there are usually lung complications, and tubercle bacilli in the sputum.

A man, aged thirty-four years, came under my care, who for six years had suffered from pain in the stomach, coming on from one to three hours after food. The pain was often accompanied by vomiting. Latterly he had been much troubled with flatulence and constipation. The patient was extremely emaciated, and his stomach was greatly dilated.

Gastric Analysis

Total acidity	12
Volatile acids	6·4
Total chlorides	0·26
Free HCl	0·00
Protein HCl	0·00
Mineral chlorides	0·26

The gastric analysis suggested cancer, but the absence of

impaired motility of the stomach, the irregular temperature, and the long history were against malignant disease.   It was thought that his condition was due to tuberculous peritonitis.   On opening the abdomen I found very enlarged mesenteric glands, and two cicatricial strictures of the small intestine, evidently the result of contraction of two tuberculous ulcers.   The strictures were short-circuited by a lateral anastomosis, and the patient was relieved of his pain and vomiting, but died about nine months later from tubercle of the lungs.

6. **Chronic Nephritis.**—Chronic nephritis may be accompanied by anæmia, vomiting, loss of appetite, and wasting to such a degree that cancer of the stomach is suggested.   The history of increased frequency of micturition, the condition of the blood-vessels, and the examination of the urine usually will reveal the true nature of the case.

SARCOMA OF THE STOMACH

Frequency.—Primary sarcoma of the stomach is a rare disease, though probably less uncommon than is supposed to be the case.   Some specimens recorded as carcinomata have been found later really to be sarcomata.   Drs. Perry and Shaw, on re-examining fifty museum specimens of gastric carcinoma, found that four were sarcomata.   Dr. Soltau Fenwick estimates that from 5 to 8 per cent. of all primary neoplasms of the stomach are sarcomata.   I think this is over-estimating the frequency of sarcomata, and I believe 4 per cent. to be nearer the correct proportion.

An important feature of gastric sarcoma is its occurrence in the early years of life.   A number of cases in children have been recorded, one by Dr. H. Thursfield in a child aged four years.

Gastric sarcoma may grow outwards, and so present itself as a tumour on the external surface of the stomach.   Such tumours are termed " exogastric " sarcomata.

Pathology.—All varieties of sarcoma have been found in the stomach. Those found most usually are round-celled sarcomata, originating in the sub-mucous tissue, and spindle-celled or fibro-sarcomata, occurring generally as circumscribed, rounded tumours of the gastric wall.

Spindle-celled sarcomata may attain a large size. Cartwell has reported an instance, in which a fibro-sarcoma of the stomach weighed twelve pounds.

Less commonly the two following types of sarcoma occur : lympho-sarcoma, commencing in the lymphatic nodules of the sub-serous coat, and myo-sarcoma, having its starting-point in the muscular wall. Unlike carcinoma, sarcoma originates usually in the body of the stomach. The round-celled variety is most malignant, and metastases are found most commonly in the liver.

Symptoms.—The symptoms of gastric sarcoma are similar to those of carcinoma. The general symptoms are most marked in the round-celled type, in which early and progressive anaemia is very common. In some instances, especially in the cases of slow-growing fibro-sarcomata, the symptoms may pass almost unnoticed. In one case which I saw, the first symptom which attracted attention was a severe haemorrhage which proved fatal within a few hours.

Dr. E. von Graff has reported the case of a young woman, aged twenty-seven, who had suffered from occasional attacks of weakness. After two years she noticed that her waist had grown larger, and found that she had lost fifteen pounds in weight. At the end of three years a large tumour had made itself apparent in the lower abdomen, reaching to the umbilicus. There was no anaemia, dyspepsia, or constipation. The tumour grew from a small segment of the greater curvature, and was removed easily by resecting a small portion of the stomach with it.

As the pylorus is affected less frequently in gastric

PLATE XLVII.
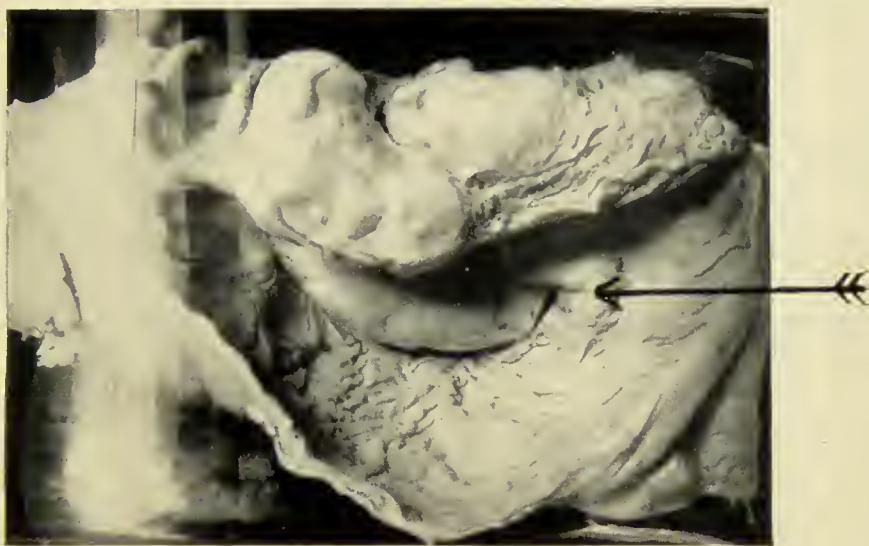
FIG. 73.—A sarcoma of the stomach, from a woman aged 22, who died with symptoms of perforation. The arrow points to the seat of hæmorrhage which was the cause of death.
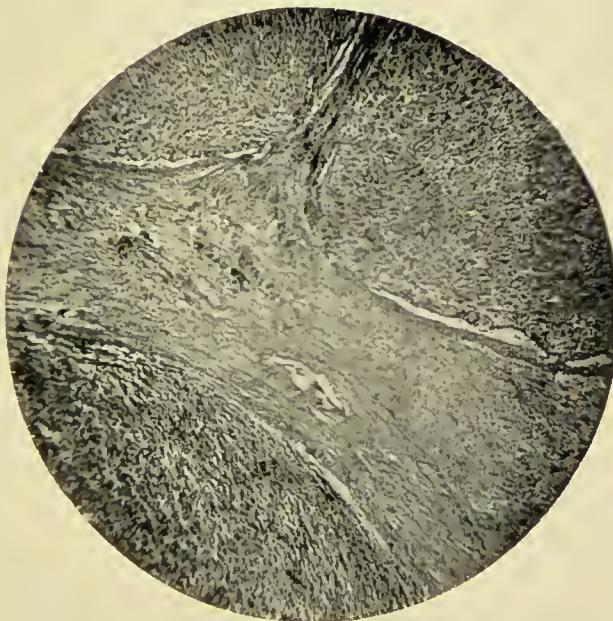
FIG. 74.—A microphotograph of the same case.

sarcoma than in gastric carcinoma, vomiting is less common. In fibro-sarcomata and myo-sarcomata, a palpable tumour almost always is present, while in round-celled sarcoma a tumour is felt less commonly.

The differential diagnosis of sarcoma from carcinoma in most cases is impossible. The younger the patient the greater is the probability of sarcoma, and early anæmia and pyrexia are in favour of sarcoma. As to the findings of gastric analysis in gastric sarcoma, I have had no experience.

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CHAPTER XVII

THE TREATMENT OF GASTRIC CARCINOMA AND SARCOMA

PROBABLY it is hardly realised yet, that although the stomach is more frequently affected with cancer than any other organ in the body, partial gastrectomy is one of the uncommon operations of surgery. This is not as it should be, for gastric carcinoma is markedly a local disease, and can be removed with favourable results, both immediate and remote, whereas no other form of treatment can hold out the slightest prospect of any lasting benefit. The good results which can be obtained in rectal or mammary cancer are well known. There is no reason why the results of surgical intervention for gastric cancer should not be as good, or indeed better, for gastric carcinoma, in its early stage, lends itself to wide removal even more favourably than cancer of the breast or rectum.

Cancer of the Pylorus.—Pyloric cancer should be treated by partial gastrectomy, whenever this is possible (p. 64).

As I pointed out in the previous chapter, the margin of induration in the stomach wall corresponds to the limit of the growth in the mucous membrane. Invariably the disease extends in the submucosa, beyond the limits of the disease in the other coats of the stomach. In partial gastrectomy, therefore, the line of section of the stomach must be made well beyond the indurated margin. In every case, at least one inch of the duodenum, the whole of the lesser curvature, together with the gastro-hepatic glands, must be removed. As to the site of section along the greater curvature, there is some difference of opinion ; some surgeons

make the section about the junction of the pyloric with the middle third, others make the section considerably further to the left.   Personally, I remove at least half of the greater curvature in early cases, and in more advanced cases I make the division close up to the isolated area, thus removing two-thirds of the stomach, together with the gastro-colic glands lying along the greater curvature.

If the glands at the lower end of the œsophagus, or round the cœliac axis, be infected with carcinoma, a radical operation is impossible, but the surgeon must not assume, because these glands are enlarged, that the enlargement is due to infiltration with carcinoma.   In these cases, therefore, it is my practice to remove one of the suspected glands for microscopical examination, then to perform gastro-jejunostomy, the resection being postponed until a later date.   If the enlargement of the glands prove to be inflammatory, the resection can be performed three weeks later.

As I have pointed out in the previous chapter, the glands along the greater curvature communicate with the glands lying in the gastro-colic omentum, along the upper border of the transverse colon.   In some cases these latter glands are enlarged.   If the enlargement be due to carcinoma, then I am of opinion that the transverse colon should be resected. I am convinced, from an examination of many specimens, that recurrence occurs, not infrequently, either in, or close to, the transverse colon, and I believe that such recurrence would be prevented, in some cases, by removal of the transverse colon with its lymphatic glands.

A patient upon whom I had performed partial gastrectomy returned to me nearly three years later, with a large mass in the upper abdomen which I took to be a recurrence. Two months later she was admitted into the London Temperance Hospital in a dying condition, with signs of general peritonitis.   At the post-mortem examination general

suppurative peritonitis was found, the result of perforation of a stercoral ulcer on the proximal side of a small, hard, ring-like stricture of the transverse colon due to a small carcinomatous growth. The large swelling felt during life consisted of a mass of matted omentum and intestine, evidently due to plastic peritonitis secondary to perforation of the stercoral ulcer. The secondary, like the primary growth, was a spheroidal-celled carcinoma. It could have been removed, so that had recognition of the true nature of the abdominal swelling been possible, the patient's life might have been saved. In this case, as in others I have seen, recurrence might have been prevented by excision of the colon. I think, therefore, the advisability of resection of the transverse colon must be considered, when the glands draining it are affected. This does not prolong the operation as much as might be expected, because, although the anastomosis takes extra time, less time is occupied in dealing with the gastro-colic omentum. At the same time, excision of the transverse colon does materially increase the risk of operation.

Personally, I am not in favour of very extensive operations as a routine treatment for malignant disease. I think that if our results are to be improved, it will be by earlier, not by more extensive, resections. In some cases, however, an extensive operation is necessary if the disease is to be removed at all. In an advanced case, the advisability of such an operation depends on the general condition of the patient. Severe anæmia is the chief contra-indication to partial gastrectomy. Such patients do not bear operation well, and usually metastatic deposits are present already. The presence of secondary growths in the pelvis is an absolute contra-indication to resection, therefore a pelvic examination should be made before deciding on surgical treatment. Patients with a very low blood-pressure are bad subjects for a severe operation. Extreme emaciation is not a bar to

PLATE XLVIII.

FIG. 75.—Carcinoma of the cardiac end of the stomach.
The dilatation of the œsophagus above the stricture
is well seen. (Royal College of Surgeons' Museum.
No. 2417.)

a radical operation. The thinness of the patient facilitates operation, and as the condition of such patients usually is the result of lack of nourishment, they bear operation better than those who are being poisoned by the absorption of toxins from the growth. Much may be done to improve patients, with gastric carcinoma, for operation, by daily lavage, and by feeding them on mutton or beef essence and milk. In bad cases the period of preparation should be at least a week or ten days. The patient often will gain two to three pounds in weight, and will be in a much better position to undergo the operation.

Cancer of the Body of the Stomach.—In certain cases of cancer forming a ring-like growth at or near the middle of the stomach (cancerous hour-glass constriction), it may be possible to excise the growth and to unite the two separated portions of the stomach. I do not favour this plan. In the endeavour to preserve sufficient of the stomach to enable the junction to be effected without tension, the surgeon is likely to remove the disease insufficiently widely. In these cases I prefer to do a partial gastrectomy and gastro-jejunostomy in the same manner as for pyloric cancer.

Cancer of the Cardiac End of the Stomach.—Partial cardiac gastrectomy for cancer of the cardiac end of the stomach, as yet, has not been performed successfully on a human being. Mikulicz has operated once for this condition, but unsuccessfully. The objections to the operation are : first, the danger of injury to the pneumogastric nerves, and secondly, the difficulty of isolating the lower end of the œsophagus sufficiently freely to anastomose it to the stomach.

Krehl, however, has demonstrated that in dogs, injury of the nerves at the lower end of the œsophagus is unattended with any impairment of nutrition, and Levy demonstrated the possibility of resecting the cardiac end of the stomach in the dead body.

To carry out such a resection, however, when a mass of malignant disease is present, is a very different matter. Mikulicz has written in optimistic vein as to the possibilities of the operation, but at present the radical treatment of malignant growth about the cardiac orifice is beyond the range of practical surgery.

The Results of Gastrectomy for Malignant Disease

A. **Immediate.**—In my Hunterian lectures in 1906 I collected a series of 436 cases of partial gastrectomy by various surgeons, with a death-rate of 28 per cent., and I pointed out that the operative results were steadily improving and that the mortality rate at that time was well under 20 per cent.

Since that time there has been a still further improvement. At the Rochester clinic during the period 1897 to 1909, partial gastrectomy was performed by Drs. W. J. and C. H. Mayo 266 times with 34 deaths, an average mortality of 12·4 per cent. During the year 1909 there were 46 operations with four deaths, an average mortality of 8·6 per cent. My own mortality rate is 14 per cent. I think we may conclude fairly, that at the present time the mortality rate is under 15 per cent., and that if patients are submitted to operation at an earlier stage, before they become exhausted by vomiting, and by poisoning from the ulcerating growth, the operative mortality need not exceed 5 per cent.

B. **Remote Results.**—The remote results of gastrectomy may be considered from two points of view; first, as to the effect of removal of all, or part, of the stomach on the health of the patient; and, secondly, as to the freedom from recurrence of the disease.

While the value of a good stomach is undoubted, fortunately Nature is able to dispense with this organ, as most of its functions can be performed vicariously by other portions of the alimentary canal. The stomach performs four main

functions. First, it acts as a reservoir, but after its complete removal the lower end of the œsophagus becomes dilated and serves this purpose, although to a limited extent. Secondly, the mechanical function of the stomach—namely, maceration—can be replaced by careful dieting. Thirdly, the secretory or chemical functions, the secretion of pepsin, hydrochloric acid, and rennin, can be replaced effectively by the intestine. Thus, the functions of the rennin can be performed by the pancreatic juice, while the antiseptic action of the hydrochloric acid, which prevents the formation of lactic acid, is carried on by the bile. Fourthly, the resorptive functions, by which peptones are absorbed, are relegated to the small intestine. Nature has thus duplicated three out of the four functions performed by the stomach, although the loss of the gastric secretion cannot be compensated altogether; " those who have lost it," says Lubarsch, " have one weapon less in the struggle for existence." Pachon and Carvalho have shown that dogs may gain in weight and remain in perfect health after removal of the entire stomach, while the elaborate observations made on Schlatter's patient, prove that the same holds good of human beings. These observations record that the absorption of albumin was unaffected, and that no putrefactive changes occurred in the intestines, which shows that the absence of the gastric juice does not lead to decomposition in the intestines.

Next as to the freedom from recurrence. In my previous book on gastric surgery I published the following table showing the after-results of total and partial gastrectomy :

	Recovered.	Died since Operation.	Still Living and Well.	
			Three Years after Operation.	Five years after Operation.
Total gastrectomy . .	17	5	6	3
Subtotal gastrectomy .	14	6	3	3
Partial gastrectomy .	55	35	12	6
. Total . . .	86	46	21	12

When recurrence ensues after partial gastrectomy, it does so usually within four years. Dr. W. J. Mayo has reported a case in which death from recurrence occurred after eight years, but this is exceptional. If a patient be free from recurrence after four years, there is a reasonable prospect of cure. Judged by this standard, 15 per cent. of the patients in this series who recovered from operation were cured. At the present time even more favourable results are being obtained. In a later report Dr. W. J. Mayo states that of the patients operated on more than three years ago, who recovered from operation, 29 per cent. have remained well. Personally I can point to one patient alive and well seven years after partial gastrectomy, and two over five years. With earlier diagnosis and earlier operation we shall obtain even better results, for there is no reason why, in cancer of the stomach, we should not reach the 50 per cent. of cures, which has been obtained by Dr. Halsted in the operative treatment of cancer of the breast.

Palliative Operations.—Opinions are divided as to the advisability of performing resection in cases in which the whole of the infected glands cannot be removed.

Under such circumstances, some surgeons are in favour of an incomplete or palliative resection for three reasons: first, because the mortality of resection is little greater than that of gastro-jejunostomy; secondly, if the patient recover from the operation, life is more comfortable and more prolonged than after gastro-jejunostomy; thirdly, as Mikulicz points out, the patient is benefited greatly by the removal of the sloughing mass of growth.

Riese follows the teaching of Mikulicz, and advises extensive resections for carcinoma, even though the disease be widespread. He believes permanent cures may be hoped for, even when there is extensive glandular disease. Let us briefly consider the propositions here stated. It is

quite true that the mortality of gastro-jejunostomy for gastric carcinoma is high. The cases in which it is performed necessarily are advanced cases. If, however, partial gastrectomy is to be adopted as the routine treatment in very advanced cases, I feel sure that the mortality would prove to be, not a little higher, but a great deal higher than that of gastro-jejunostomy. Whether life is prolonged appreciably by a resection which leaves disease behind, is open to doubt; at any rate the gain is not great, and if the increased mortality rate be taken into account, I question whether, on the balance, resection would show an increased gain in life as compared with gastro-jejunostomy. There is room for difference of opinion as to whether patients are more comfortable after a palliative resection. Doubtless the majority are, but many patients, after a gastro-jejunostomy, live in comfort almost to the last.

As to the statement, that the removal of the sloughing mass of growth greatly benefits the patient, I would reply that a sloughing mass is not present in all cases. The benefit derived from a palliative resection will depend on the type of the growth. With a hard, constricting, slow-growing carcinoma of the pylorus, the patient suffers from starvation more than from toxæmia; with a soft, fungating growth toxæmia is a prominent symptom. In the latter cases patients are undoubtedly much benefited by the local removal of the growth.

It may be argued, that as palliative resection of a cancerous ulcer of the tongue, the inoperable glands being left, is a very beneficial proceeding and is considered to be good surgery, so palliative gastrectomy should be performed for the same reason. There are, however, two important differences in the two cases. In cancer of the tongue, pain is a constant and distressing symptom. In gastric carcinoma, apart from obstruction, which can be relieved by

gastro-jejunostomy, this is not so. The relief afforded by a palliative resection is much greater in lingual than in gastric carcinoma. Secondly, local removal of a portion of a cancerous tongue, the glands being left, is comparatively safe; partial gastrectomy in an advanced case is a very serious operation.

At this stage let me point out that wide resection may be performed for two totally distinct reasons. A wide resection may be done in an early case, in order to make certain of removing all the microscopic ramifications of the disease. On the other hand, a very extensive excision may be absolutely necessary in order to extirpate all the macroscopic disease. These two classes of gastrectomy should be distinguished clearly. Both the immediate and remote results are in marked contrast. A wide resection in an early stage of the disease is attended with a low mortality rate, and with good remote results. Extensive operations in advanced cases have a high death-rate, and the ultimate results are disappointing.

The advocates of palliative resection may argue, that even in an early case it is possible that, however wide the resection, some cancer-cells are left behind. Mr. Sampson Handley has shown, that carcinomatous cells permeate the lymphatic channels very widely from the primary growth. Fortunately, the clinical results of resection are vastly better than pathological teaching leads us to expect. We must, therefore, suppose that Nature, in some measure at least, is able to deal with the cancer-cells which are left behind. If this were not so, resection, even in an early case, would be a futile proceeding, if we accept as true all that pathologists tell us.

To what extent, and under what circumstances, Nature does this, we are in the dark. We have, however, no evidence that if infected glands be left behind, permanent cure can result.

This being so, my view is, that if there be inoperable carcinomatous glands, it is not worth while to submit the patient to the risk of an extensive resection, the benefit of which cannot but be short-lived. Before deciding against resection in a given case, the surgeon must ascertain by microscopical examination whether the enlargement of the irremovable glands is due to cancer or to simple inflammatory infiltration.

Even in those advanced cases in which it may be possible to remove all the disease by means of a very extensive operation, the advisability of resection, I think, is doubtful.

In early cases I am an advocate of resections which are wide in comparison with the extent of the disease, but I am not so sure of the benefit resulting from resections which have to be extensive on account of the advanced stage of the disease. The surgeon ought not to have to consider this problem, and it would be seldom necessary to do so if the stomach-tube, and other available means of diagnosis, were resorted to in the routine of practice. Early diagnosis must be our aim, and it is by realising this ideal, and not by the performance of more elaborate and extensive operations in advanced cases, that the results of the surgical treatment of gastric carcinoma will be bettered.

In cases of pyloric cancer, with gastric stasis and food retention, too advanced for resection, a gastro-jejunostomy should be performed.

The increased nutrition, and relief from vomiting, greatly benefit the patient. The operation, however, should not be advised if there be ascites, or marked hepatic enlargement. Under such circumstances the risk of the operation is so great, and the prospect of prolongation of life so small, that it is better to treat the stenosis by daily gastric lavage. Unless there be food retention, I do not think that a short-circuiting operation is of any benefit.

In those cases in which the whole of the stomach is infiltrated with cancer, so that a gastro-jejunostomy is impracticable, the advisability of performing a jejunostomy may be considered. The advantages of this operation are very doubtful. It is not to be recommended except in a few exceptional cases, in which there is some urgent reason for making an attempt to prolong the patient's life for a few weeks. Under ordinary circumstances the benefit to be derived from it is so slight, that it is not worth doing.

In cancer of the cardiac end of the stomach, or of the œsophagus, gastrostomy is the only surgical procedure available. In my judgment, it should not be advised until the patient no longer is able to swallow sufficient nourishment to maintain his body weight. I do not agree with the teaching that gastrostomy should be performed as soon as the diagnosis has been made. If this practice be followed, it is almost certain that in some cases, the operation will precede the cancer. I have seen patients who have had a gastrostomy performed and who have refused to die, because they have been the victims, not of cancer, but of an erroneous diagnosis.

In hard cancerous strictures of the œsophagus the use of a Symonds' tube (Fig. 76) often is very valuable. This

FIG. 76.—A Symonds' Tube.

instrument is less used than its merits deserve. Why, I do not know. If it be passed by the aid of a string guide as suggested by Dr. Plummer, it is as safe as gastrostomy, and in many cases is preferable. In the soft fungating growths its use is attended with greater risk, but even in these cases its use for a few days often will enable the surgeon to feed

up a starved patient and bring him into a better condition for operation.

Patients with cardiac or œsophageal cancer die more often from broncho-pneumonia and metastases than from starvation. In a few selected cases gastrostomy prolongs life and adds to the patient's comfort. The operation, therefore, has its uses, but the number of cases in which it is of marked benefit is limited.

The immediate operation mortality is very small, probably about 2 or 3 per cent.

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CHAPTER XVIII

ULCUS CARCINOMATOSUM

OF late years there has been considerable discussion as to the frequency with which cancer is grafted on simple ulcer of the stomach. Cruveilhier, in 1839, and Dittrich, in 1848, were the first to point out this possibility.

Zenker, in 1882, went further, and put forward the hypothesis that *all* cases of gastric carcinoma have their origin in simple ulcer. More recently Drs. W. J. Mayo and Christopher Graham have laid great stress on the origin of cancer in ulcer, and Drs. Wilson and MacCarty from the same clinic, have contributed a valuable paper dealing with the problem from a pathological standpoint. These writers state that they have been able to demonstrate a pre-cancerous or non-malignant ulcer in 62 per cent. of their cases. Most observers, however, have placed the proportion at a much lower figure. Thus, according to Sonicksen, 14 per cent. of all cases of cancer of the stomach originate in ulcers ; Lebert estimated the frequency at 9 per cent. ; Rosenheim at 6 per cent. ; Fenwick and Häberlin at 3 per cent.

The possibility that carcinoma may originate in an ulcer cannot be denied ; however, it is very doubtful whether such an occurrence is as frequent as some authorities would have us believe. The problem is a very difficult one, and a dogmatic conclusion impossible, but the evidence available in favour of the view that simple ulcer is a frequent pre-cursor of cancer is not convincing. This evidence is both clinical and pathological.

(*a*) **Clinical Evidence.**—A number of the patients who come under observation with gastric carcinoma give a history of gastric trouble extending over many years. The first step in the argument is the assumption, that the gastric symptoms are due to gastric ulcer. When, subsequently, on the operation-table, or in the post-mortem room, the existence of cancer is confirmed, the argument is carried a stage further by assuming, that the cancer has originated in the ulcer, which, from the history, is supposed to have existed previously.

The argument may be summarised briefly thus : Cancer is a disease, the duration of which is comparatively short ; a long history of gastric trouble is evidence of the presence of an ulcer ; therefore, when cancer is associated with a long history, the cancer must have been grafted on a simple ulcer. Such an argument is unscientific. It contains two fallacies, and disregards an alternative conclusion, at least of equal probability.

The first fallacy is, the assumption that we can diagnose a gastric ulcer from the clinical history alone. In duodenal ulcer, it is true, the history often is so typical, that from it a diagnosis may be made with confidence. This is not so with gastric ulcer. Usually the diagnosis of duodenal ulcer is easy, that of gastric ulcer very difficult. From operative experience we have learnt that gastric ulcer is far less common than formerly was supposed. The symptoms attributed to gastric ulcer frequently are due to disease of the appendix, or of the gall-bladder, to septic gastritis, or to intestinal toxæmia. Although in some instances, when dealing with a particularly intelligent patient, it may be possible from the history alone to differentiate between these conditions, in the majority of cases it is not so. I maintain, therefore, that because a patient with cancer of the stomach gives a long history of dyspeptic trouble, we are not justified in

assuming that the symptoms have been due to a gastric ulcer. The second fallacy is the assumption that the duration of carcinoma is never protracted. It is conceivable that the growth of cancer in its early stages is much slower than we think. At present we have no means of determining the life-history of cancer of the stomach. We do know, however, that in some instances it is of slow growth, and that dissemination is a late manifestation. Most surgeons have met with instances, in which, after gastro-jejunostomy for inoperable cancer of the stomach, the patient has lived for years before succumbing to the disease.

Some years ago I performed gastro-jejunostomy on a woman, aged twenty-seven years, who had an extensive pyloric cancer with such widespread glandular infection, that a radical operation was impossible. A year later she had gained nearly three stone in weight, and was so well, that when the American Society of Clinical Surgery was in this country, I showed her to the members as a case in which I had been mistaken as to the nature of the disease. The patient remained well for two years, then exhibited signs of sudden, rapid growth of the disease, and died within a few months.

A more remarkable case is that of a man, aged fifty-two years, upon whom I performed gastro-jejunostomy in 1908. He had cancer of the pylorus, which had spread in the submucous tissue almost up to the cardiac end of the stomach. Radical operation was impracticable. I removed a portion of the growth, and microscopically it proved to be a spheroidal-celled carcinoma. It is now more than $4\frac{1}{2}$ years since the operation, and the patient states that " he is as well as he has ever been in his life." Until we know more of the natural history of gastric cancer, we are not justified in drawing conclusions as to whether life is, or is not, greatly prolonged in some instances.

The alternative to the conclusion that carcinoma is grafted on ulcer is, that simple ulcer and carcinoma may occur independently in the same patient. That sometimes this should happen is highly probable, for the frequency of the incidence of cancer and ulcer is very similar. Further, it is a hypothesis based on fact. On several occasions, when operating for cancer, I have been able to demonstrate to those present, either an open ulcer, or the scar of an ulcer, quite distinct from the malignant growth. In several, at least, of the cases recorded as instances of ulcus carcinomatosum, two ulcers were observed, one simple and the other malignant.

A perusal of the records of cases of ulcus carcinomatosum reveals one striking feature, namely, the frequency with which there is an interval between the symptoms of supposed ulcer and the onset of the symptoms of gastric cancer. With regard to such cases as these, the assumption that cancer has been grafted on an ulcer is less reasonable than the hypothesis, that an ulcer has been present and has become healed, and subsequently cancer has occurred independently of the ulcer.

(b) **Pathological Evidence.**—According to Drs. Wilson and MacCarty, in 71 per cent. of their series of cases there was " a large ulcer with scar tissue centres and overhanging borders, deep in the bases of which cancer is present, which ulcer in almost every instance has unmistakably originated in the lesser curvature of the stomach, the usual site of gastric cancer. Further, almost every case gives a clinical history suggesting gastric ulcer for a long period of years preceding the relatively short period when the history became that of gastric carcinoma." Apart from the assumption that a previous history of gastric trouble is evidence of gastric ulcer—an assumption which I have criticised already —the weak point of this argument appears to me to be

absence of proof that an ulcer in which cancer cells are present, has ever been other than malignant. The presence of scar tissue is not necessarily evidence of non-malignancy. We know that in cancer of the breast there may be formation of scar tissue, Nature's attempt at arrest of the disease. Further, the border line between a benign and a malignant growth is a narrow one, and a given tumour may be inherently malignant before, clinically or pathologically, we can recognise its malignant character, with the means at present at our disposal.

There are two truths which appear to me to make it very difficult to accept the view, that the grafting of cancer on ulcer is of frequent occurrence. First, simple ulcer of the duodenum is at least as common as gastric ulcer, and yet cancer of the duodenum is a rare disease. Surely, if simple ulcer be a frequent precursor of cancer, then cancer of the duodenum should be at least as frequent as gastric cancer. Then again, cancer is commoner in the second than in the first portion of the duodenum. Ulcer in the second portion is the rare exception. If ulcer be the precursor of cancer, then cancer should be far commoner in the first than in the second portion. Exactly the contrary is the case.

Secondly, death from cancer, after gastro-jejunostomy for supposed simple ulcer, is a rare event. If the hypothesis as to the frequency with which simple ulcers become malignant be correct, then many of the ulcers supposed to be simple should prove later to be malignant, and consequently, many of the patients on whom gastro-jejunostomy is performed, should die subsequently from cancer. As a matter of fact, the mistake, when made, is more often in the opposite direction; it is the supposed malignant growth which proves to be innocent, not the supposed simple ulcer which proves to be malignant. My own experience is that only one per cent. of the patients on whom gastro-jejunostomy is per-

formed for simple ulcer, die later from carcinoma. Professor Kocher has published recently, a review of fifty cases of gastro-jejunostomy. The period which had elapsed since operation varied from two to twelve years. In no case was there a recurrence of symptoms, or any suggestion of malignancy. Dr. Gressot states that malignant degeneration of surgical ulcers occurs in only 2·3 per cent. of all cases after gastro-jejunostomy.

In view of such observations it is difficult to accept the view, that grafting of cancer on simple ulcer is a frequent event. Of course I do not deny that a simple ulcer may become malignant. My point is, as regards the frequency of such a happening. That it is anything like so common as some authorities teach, in my opinion is doubtful. The subject requires much further investigation. In the meantime the verdict should be, the Scotch verdict of " Not proven."

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CHAPTER XIX

INFANTILE HYPERTROPHIC PYLORIC STENOSIS

I use the term " infantile " in preference to " congenital "
as the hypothesis, that the disease exists before birth, has
not been verified. It is only within recent years that the
disease has been recognised clinically. According to Sir
William Osler, the earliest record of a case is that described
by Hezekiah Bardsley in the year 1788, under the title of
" A Case of Scirrhus in the Pylorus of an Infant." Additional
cases were reported by Williams in 1841, and Daworsky in
1842, but these isolated records were forgotten until the
year 1888, when Hirschsprung drew attention to the sub-
ject anew.

It is likely that the disease is more common than re-
corded cases and museum specimens would lead us to
believe. Probably many of the patients suffering from the
disease are treated without the true cause of the symptoms
being ascertained.

Morbid Anatomy.—The essential feature in all recorded
cases and museum specimens is a marked thickening of the
pylorus. This thickening is due to a hypertrophy, or rather
hyperplasia of its circular muscular fibre, without any
evidence of inflammation. The mucous membrane lining
the pylorus is thrown into folds. Dr. Cautley and Mr. Dent,
in a valuable paper, point out that a single reduplication of
the mucous membrance, much more marked than any other
fold, is a conspicuous feature in many of the specimens
(Plate XLIX, Fig. 77). Both these writers and Dr. John

PLATE XLIX.
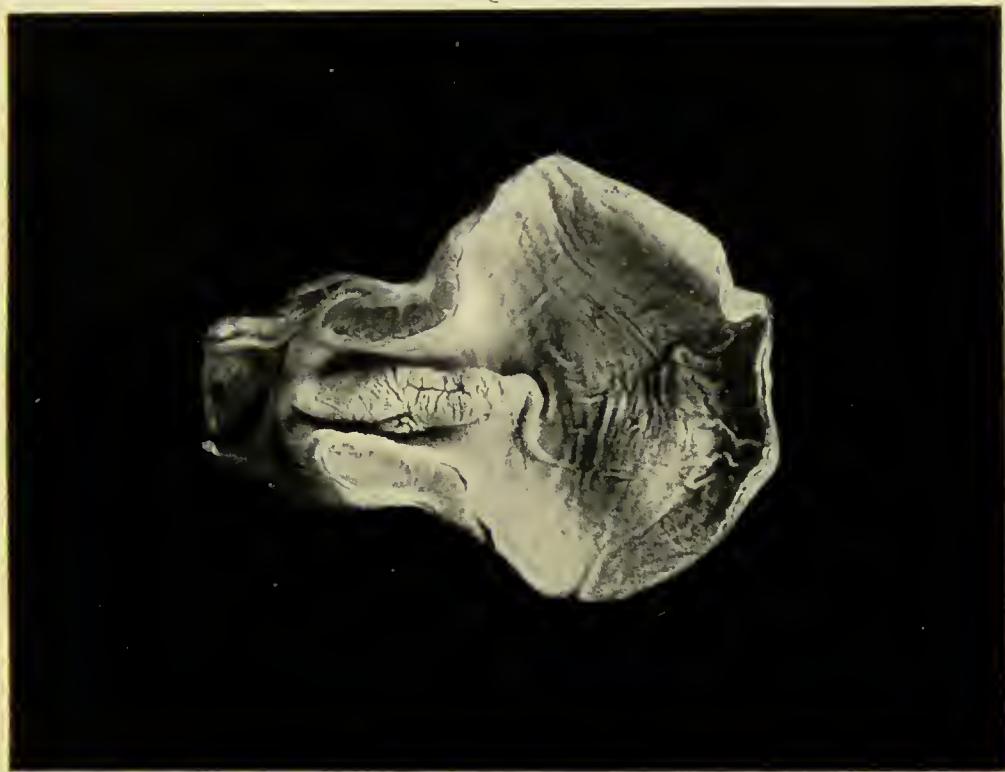
FIG. 77.—Infantile pyloric stenosis.   The fold of mucous membrane is well seen.
(Royal College of Surgeons' Museum.   No. A2398a.)

Thomson agree that as a rule there is no thickening of the submucous coat. The thickening is usually more marked at the duodenal end of the pylorus, is often of cartilaginous hardness, and in length measures about one inch. Stenosis is not complete, for a probe can be passed readily through the pylorus, but during life obstruction probably is complete owing to the folds of mucous membrane described above.

Two curious features of the disease are, its frequency in male infants, and the relatively large proportion of first-born children who are affected. About 80 per cent. of all cases occur in males.

Ætiology.—The ætiology of the disease is uncertain. Two hypotheses have been suggested :

1. That the thickening of the pylorus is a definite anatomical condition due to hyperplasia or hypertrophy of the muscular tissue. Some authorities regard the hyperplasia as primary, while according to others the increase of muscular tissue is of the nature of a hypertrophy secondary to spasm of the pylorus.

2. That the symptoms are not the result of an anatomical lesion, but of a functional spasm of the pylorus.

These views may be represented graphically thus :

Infantile Stenosis $\begin{cases} 1. \text{ Anatomical} & \begin{cases} a. \text{ Primary hyperplasia.} \\ b. \text{ Secondary hypertrophy.} \end{cases} \\ 2. \text{ Functional} & \quad \text{Spasm of pylorus.} \end{cases}$

At the present time the available evidence is in favour of the view that the condition is a primary local overgrowth— in other words, a freak of Nature. Dr. Cautley, who adopts this view, says that " Nature in her anxiety to provide an efficient pyloric sphincter has over-exerted herself, and produced too large a quantity of muscular tissue." In support of this view, it has been urged that when a child, affected with infantile pyloric stenosis, dies within a few weeks of

birth, the amount of hypertrophy found is greater than could have been produced since birth (Rolleston). If the pyloric hyperplasia existed at birth, it must have been either a primary developmental abnormality, or due to spasm set up by the swallowing of amniotic fluid *in utero*. That a fœtus swallows amniotic fluid is at present pure assumption.

As against the view that the hyperplasia is primary, it is urged that hypertrophic pyloric stenosis never has been observed in a dissected fœtus. On the other hand, the occurrence of the disease in three members of the same family, as recorded by Henschell, at least suggests the possibility of a congenital origin.

Symptoms.—While authorities differ as to the ætiology of the disease, there is striking unanimity as to the clinical symptoms.

Usually the infants affected are healthy-looking babies of normal weight. The prominent symptom is vomiting. Within a short period, varying from a day or so to three or four weeks, the infant begins to vomit after its feeds. The vomit consists of altered food with a little mucus. Bile is not present. Change of milk has little or no effect on the vomiting. Dr. Robert Hutchison has pointed out a curious feature of the vomiting, namely, its tendency to cease for some hours with any change of food, and to recur again as violently as ever. At first the quantity vomited is small, and some feeds may be kept down, and later apparently the whole may be brought up at once. Gradually the vomiting increases in frequency and amount, and is often forcible in character, while the infant's weight steadily decreases, and its face becomes pinched and wizened.

The condition is distinguished from the gastro-enteritis, so common in children, by the absence of diarrhœa ; as little or no food passes through the pylorus, there is little fæcal

residue to be evacuated, and so obstinate constipation is a marked symptom.

In the early stages of the illness, little can be made out from an examination of the abdomen, but later the stomach becomes dilated; the intestine being empty, the lower part of the abdomen is collapsed and hollowed out, so that the contrast between the lower part of the abdomen and the upper part, distended by the dilated stomach, is very characteristic.

At a later stage, the outline of the stomach may be made out definitely, and waves of contraction can be seen passing across the upper abdomen from left to right. In some instances a definite pyloric tumour may be felt, especially if the examination be made under an anæsthetic—a precaution which should not be omitted in a doubtful case.

Treatment.—There is little doubt that some patients recover without surgical intervention. Dr. G. F. Still has had under his care several undoubted cases, in which recovery has ensued after daily lavage of the stomach. In all of his cases there was a dilated stomach and a definite pyloric lump. In a case recorded by Dr. F. E. Batten, the diagnosis was verified subsequently by post-mortem examination, as the child, after gaining $8\frac{1}{2}$ pounds in weight in five months, succumbed three months later to an attack of gastro-enteritis and pneumonia. Dr. Hutchison records 18 recoveries in a series of 20 cases. Heubner has reported 21 cases treated medically with 19 recoveries, Starck 12 cases with 11 recoveries, Bloch six cases with recovery in all of them, and Bendix 32 cases with recovery in 30. I myself have seen two patients recover with regular daily lavage.

These facts should make us pause before advocating operation. Further, it is important to note, that in the cases which have been treated successfully without operation, vomiting has diminished and finally ceased, but only after some weeks of regular treatment.

Therefore a very thorough and extended trial should be given to careful feeding and regular lavage, before resorting to surgical measures. It must be admitted, that in hospital practice this treatment requires so much individual attention, that it is difficult to carry it out, while the impossibility, in an institution, of feeding the children by the breast, is a serious drawback; but in private practice, if the mother be unable to nurse the child, the services of a wet nurse can be requisitioned, in addition to the undivided attention of a skilled nurse.

It is noteworthy, that in many of the cases which have been treated successfully by Loreta's operation, vomiting has not ceased immediately, and the feeding of the infant has required much care and patience for some time. Had the patience been exercised before operation, possibly the necessity for operation might not have arisen. Present experience, then, seems to indicate that if these cases be recognised early, treated by systematic lavage, and fed by the breast, there is at least a possibility of recovery. Whether this recovery is absolutely permanent, time alone can show. At any rate, if operation can be delayed until the infant be older and stronger, something will have been gained.

Schwyzer, in 1897, was the first to propose, and Meltzer, in 1898, the first to practise, surgical intervention for this disease. Four different procedures have been adopted: namely, pylorectomy, gastro-jejunostomy, pyloroplasty, and pylorodiosis (Loreta's operation).

Pylorectomy may be dismissed at once as an unsuitable operation for such young patients. Mr. H. J. Stiles, who, acting on the opinion expressed by Meltzer, performed the only operation recorded, condemns it as unnecessarily severe and unjustifiable.

Gastro-jejunostomy.—The first successful gastro-jejunostomy for this disease was performed by Löbker in July 1898.

So far the mortality attending this operation in these cases has been very high, but it is only fair to point out that many of the little patients were very emaciated and in an extremely unfavourable condition for operation.

In most of the cases the posterior method was adopted, but my own opinion is, that in these very young patients, the anterior operation is attended with less risk. In my Hunterian lectures I recorded cases in which the patients were in excellent health six years after operation.

Pylorodiosis.—The mortality of pylorodiosis also is high. There is considerable risk of rupturing the duodenum ; on the other hand, if the stretching be not sufficient, it is extremely likely that relapse will ensue.

Pyloroplasty.—The first pyloroplasty was performed by Braun. In some instances the operation has proved very satisfactory, but in others there has been trouble with the feeding—a serious matter when the condition of the child before operation is considered.

The Choice of Operation.—So much turns on the condition of the infant at the time of operation, that it is impossible to estimate the relative mortality of these different procedures, and probably it is on this factor and on the skill of the operator, rather than on the particular operation adopted, that the immediate result depends.

The one thing certain is, that surgical intervention in these cases is a serious matter. Whatever operative method be chosen, the mortality rate is high. Therefore, every effort should be made, in the direction indicated above, to obviate the necessity for operation. On the other hand, we must guard against delay, until the child be in such condition that recovery is almost hopeless, as has happened in many of the recorded cases. Pylorodiosis appears to be the least severe procedure, but the after-results hardly can be considered satisfactory. It is only fair, however, to state that some

surgeons have obtained satisfactory after-results by this operation. Mr. Burghard tells me that he has had only three instances of recurrence of symptoms in the twenty-five patients who recovered after operation. In two of the cases, a second operation was necessary; in the third case, the patient recovered with lavage.

The mortality rate of all the procedures available is so high, that it is difficult to decide which is the best. My own view is that the choice lies between pyloroplasty and gastro-jejunostomy.

Pyloroplasty has been advocated warmly, and supported ably, by Cautley and Dent. They maintain that it is a less severe operation than gastro-jejunostomy, and that it can be performed through a small incision, and that it brings about as complete a recovery as gastro-jejunostomy. To this I would say, that pyloroplasty is not always so easy as its advocates would have us believe. Even in an adult, it is difficult to be certain as to the size of the channel left after suturing, and this difficulty is much greater in an infant; it is significant, that in two of the recorded cases complete pyloric obstruction resulted from the operation, a complication which I think is likely to occur in a considerable proportion of the cases.

With our present knowledge it is difficult to decide whether pyloroplasty, or gastro-jejunostomy be the better operation. Whether the remote results will prove as satisfactory after pyloroplasty, as after gastro-jejunostomy, we do not know. I agree with Mr. Stiles, that in principle gastro-jejunostomy is the better operation, and the following are my reasons:

1. It is preferable to operate on normal than on morbid tissues.

2. Feeding can be commenced at once after gastro-jejunostomy, no small advantage in these cases, whereas in

the cases of pyloroplasty the feeding has caused no little anxiety.

3. If the anterior operation be performed, it can be completed in twenty-five minutes, which is but little, if at all, longer than the time required for pyloroplasty.

4. The incision need be no longer than that required for pyloroplasty ; $2\frac{1}{2}$ inches is quite sufficient. If the patient's shoulders be raised, there is no fear of protrusion of the intestine.

5. The remote results of gastro-jejunostomy in these cases are excellent. We do not know yet that those of pyloroplasty are as good.

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CHAPTER XX

FIBROMATOSIS OF THE STOMACH

THE term "fibromatosis" of the stomach has been suggested by Professor Alexis Thomson, to denote the condition which has been described variously as cirrhosis, linitis plastica, and fibroid induration of the stomach.

Pathology.—Aschoff regards cirrhosis as a sequel of chronic catarrh, the fibrous thickening extending from the mucosa to the outer coats of the stomach. Nothnagel expresses the view that it originates in, and spreads inwards from the peritoneal coat. Bret and Paviot maintain that the lesion always is cancerous, basing their conclusions, not on the microscopical examination of the submucosa, but on the condition of the perigastric lymphatic glands, and on the presence of metastatic deposits. Andral, Cruveilhier, Brinton, Wilson Fox, and Wilks take the contrary view—namely, that the lesion is not malignant. These authorities regard it as a primary cirrhosis of the submucosa, inflammatory in nature, and analogous with cirrhosis of the liver. Hitherto the view generally accepted has been that the thickening in the submucous coat is a form of atrophic, infiltrating, scirrhous carcinoma.

Professor Alexis Thomson, who, in conjunction with Dr. James M. Graham, has published recently an admirable paper on this subject, has adduced conclusive evidence that fibromatosis of the stomach may occur without the presence of cancer. These writers point out that fibromatosis may be limited to a portion of the stomach—

localised fibromatosis — or it may extend throughout the entire stomach—**diffuse fibromatosis**. Of these the localised form is not only the more frequent, but is the more difficult to differentiate from cancer.

Localised fibromatosis originates almost always in the neighbourhood of the pylorus and spreads towards the cardia, usually, but not invariably, showing a preference for the lesser curvature (Thomson). The affected portion of the stomach is densely hard and rigid, whereas the rest of the stomach is flaccid and collapses readily. Usually the glands along the curvatures are enlarged and harder than normal.

In the paper referred to, the authors state that fibromatosis is associated invariably with ulceration of the gastric mucosa, and they suggest that the " necessary antecedent of fibromatosis is a breach of the surface which permits of the access to the subjacent submucosa of a toxin or irritant, to which the tissues of the submucosa react, and the reaction takes the form of a diffuse spreading, new formation of young fibrous tissue." The formation of fibrous tissue may extend both to the mucous layer and to the serous coat.

The view that fibromatosis is due to syphilis or tubercle is not supported by convincing evidence. The characteristic granulation tissue and endarteritis of syphilis are absent. In tuberculosis of the stomach the granulation tissue with giant cells is most marked in the subserous and not in the submucous coat as is the case in fibromatosis.

In diffuse fibromatosis the whole stomach is affected —the so-called " leather bottle stomach." Such a stomach is diminished greatly in size, does not collapse when it is opened, and its capacity is much reduced. As a rule the thickening is most marked about the pyloric end of the viscus, and gradually tapers off towards the cardiac end.

Some years ago I came to the conclusion that the specimen of leather-bottle stomach (No. 2408) in the Museum of

the Royal College of Surgeons, which has been figured so often as an example of leather-bottle stomach, the result of malignant disease, would prove to be an example of fibrosis (Plate L, Fig. 78). Recently Mr. S. G. Shattock has been good enough to re-examine the specimen, and he reports that microscopical sections show no evidence of malignancy.

There is no doubt, however, that a small, hard, contracted stomach may result from diffuse infiltration with carcinoma. I have seen two examples of the condition, in which the whole stomach was invaded by cancer cells, and in both cases death resulted from dissemination.

The Differentiation of Fibromatosis from cancer of the stomach is often very difficult. The white appearance of the peritoneal coat, and the presence of enlarged glands, are extremely suggestive of carcinoma.

A number of specimens figuring in museums as cancer, and which have been removed as such, are probably examples of fibromatosis. Professor Alexis Thomson recently has re-examined seventy-two specimens labelled as cancer, in the University of Edinburgh Museum, and found that in 26·3 per cent. of them the lesion was not cancerous.

There are three points of difference between fibromatosis and cancer, which usually can be made out plainly, if a section be made through the stomach wall.

First, in cancer the differentiation of the layers of the gastric wall is less distinct than in fibromatosis.

Secondly, in cancer the infiltration is mainly in the mucous coat; in fibromatosis, in the submucous coat.

Thirdly, in cancer, as a rule, there is an ulcerated, fungating mass in the interior of the stomach; in fibromatosis no such growth is present, although usually there is an erosion or a punched out ulcer.

Treatment.—In the majority of cases in which surgical

PLATE L.
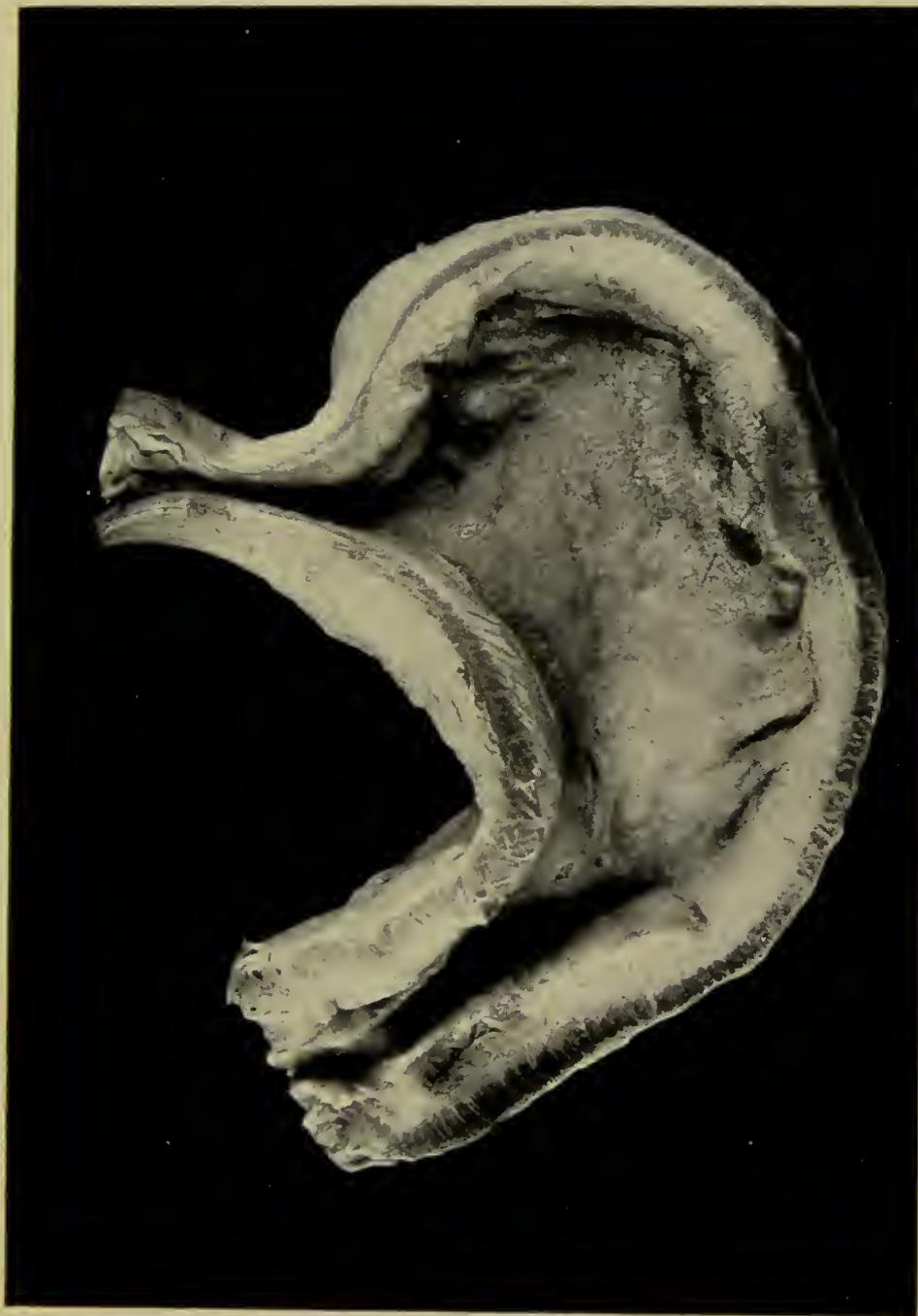
FIG. 78.—Fibromatosis of the stomach. The specimen was described hitherto as leather-bottle stomach from carcinoma. (Royal College of Surgeons' Museum. No. 2408.)

treatment has been adopted, resection has been performed in the belief that the disease was malignant. Probably a gastro-jejunostomy would give relief, but I have been able to find the record of only two cases in which this treatment has been adopted.

Professor Roux of Lausanne performed an anterior gastro-jejunostomy on a man aged thirty-two years; the patient was quite well three and a half years later. Dr. H. M. Lyle performed a posterior gastro-jejunostomy on a woman aged forty with a completely successful result. Professor von Eiselsberg has recorded a case of localised fibromatosis for which he performed jejunostomy; six years later the patient was enjoying good health.

It must be admitted, however, that on the operation table the differential diagnosis sometimes presents considerable difficulty. As Professor Thomson has pointed out, the stomach may become adherent to neighbouring organs, which may become infiltrated, thus increasing the resemblance to cancer.

In cases of doubt the best plan seems to be to perform gastro-jejunostomy at the extreme cardiac end of the stomach, and at the same time to remove one of the enlarged glands and a small portion of the stomach wall, at the pyloric end, for microscopical examination. If the tissue prove to be cancerous, resection may be performed three weeks later.

The important point with regard to these cases is, that gastrectomy should not be performed until the diagnosis is certain, with the possible exception, that when, in a young patient in good condition, there is an induration limited to the pylorus which can be drawn well outside the abdomen, a limited resection may be performed.

It must be borne in mind that a negative microscopical examination does not preclude the existence of cancer. Notwithstanding this, I do not think resection justifiable

except under the conditions indicated above. In cases of doubt the disease is more likely to prove inflammatory than malignant, and therefore gastro-jejunostomy is the safer procedure, and in the majority of cases likely to give the patient complete relief.

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CHAPTER XXI

BENIGN TUMOURS OF THE STOMACH

BENIGN tumours of the stomach are exceedingly rare.
The following varieties have been described :

1. Adenomata.
2. Myomata.
3. Fibromata.
4. Lipomata.
5. Lymphadenomata.

Benign tumours of the stomach may :

(a) Grow towards the interior of the stomach, and project into its cavity (intra-gastric tumours, polypi).

(b) Remain encapsuled in the wall of the stomach.

(c) Grow towards, and project on, the external surface of the stomach, to which they may be connected by a pedicle (exo-gastric tumours).

1. **Adenomata** are seen as rounded or lobulated projections from the interior of the stomach. They may be single or multiple, and are commonly found near the pylorus, attached to the gastric wall either by a broad base or by a narrow pedicle. They undergo cystic degeneration readily, and often are described as mucous polypi. They consist of a central core of connective tissue containing blood-vessels and lymphatics, over which is spread a thin layer of unstriped muscular tissue. They are covered with greatly hypertrophied mucous membrane.

Cases are on record in which adenomata have caused

pyloric obstruction. Some of the cases recorded as adenomata were probably adeno-carcinomata.

2. **Myomata** of the stomach, first described by Morgagni, occur as smooth, hard, round swellings in the gastric wall. They may grow either into the cavity of, or towards the outer surface of the stomach, and may become pedunculated. Occasionally they attain to a large size, and are recognised as abdominal tumours during life. Von Erlach removed successfully a tumour weighing thirteen pounds from the front wall of a woman's stomach, to which it was attached by a pedicle. Professor Von Eiselsberg removed a large fibromyoma, the size of a man's head, which was attached to the greater curvature. The patient recovered.

Deaver and Ashurst have collected forty cases of gastric myoma. About half of them were growing from the external surface of the stomach. In fourteen recorded cases of gastric myoma, secondary deposits were found in four instances—" leio-myoma malignum."

3. True **fibro-myomata** are exceedingly rare ; tumours so described usually have proved to be fibro-sarcomata. Dr. Soltau Fenwick states that there is " not a single case in the whole of the literature where a large fibroid tumour of the gastric wall was above the suspicion of malignancy." Mr. Walter Spencer, however, has described a pure fibroma which he removed with success.

4. **Lipomata** occur in the form of the lobulated tumours characteristic of lipomata elsewhere. They originate, either in the submucous layer and project into the cavity of the stomach, or in the subserous layer and grow from the external surface of the stomach. In either situation they may become pedunculated.

5. **Lymphadenoma** of the stomach is a very rare condition. The mucous membrane of the stomach is thrown into polypoid folds, affecting chiefly the middle and cardiac ends

PLATE LI.

FIG. 80.—Pedunculated fibroma attached to the edge of the pyloric orifice. (Soltau Fenwick.)

FIG. 79.—Mucous polypi in the stomach. (Royal College of Surgeons' Museum.)

of the stomach. The growths consist of lymphoid tissue. The mucosa overlying the growth may become infiltrated and ulcerated.

Lymphadenoma of the stomach is rare before middle life, but a case occurring in an infant eighteen months old has been recorded by Drs. Rolleston and Latham.

Symptoms and Treatment.—As a rule, benign tumours of the stomach give rise to no symptoms, and seldom are recognised during life. The intra-gastric tumours, if situated near the pylorus, *may* cause obstruction, and lead to such emaciation that the patient is supposed to be suffering from malignant disease.

Occasionally, benign tumours attain to such a size that they are palpable on examination of the abdomen : the chief, often the only symptom, in such cases, is abdominal discomfort. Owing to their mobility, the exo-gastric tumours, when of large size and attached to the stomach by a pedicle, may give rise to difficulty in diagnosis.

On several occasions gastric myomata in women have been diagnosed as ovarian adenomata (Maylard, Herman, Cantwell, Eiselsberg).

When single, benign tumours are amenable readily to surgical treatment. The important point is, that their benign character may not be recognised, and that partial gastrectomy may be performed, when it would be sufficient to shell out the tumour. Thus Von Hacker performed partial gastrectomy, and Hahn pylorectomy, in the belief that they had to deal with sarcomata, whereas subsequent examination demonstrated that the tumours were benign.

It is said that these tumours, if untreated, ultimately become malignant. It is more probable, that from the first, such tumours are sarcomata of slow growth.

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CHAPTER XXII

APPENDICULAR GASTRALGIA

THAT there are cases in which disease of the appendix mimics gastric disease, in some cases with very remarkable fidelity, does not admit of doubt. This does not appear to have obtained general recognition in this country, although abroad it has been recognised for some years. Twelve years ago Ewald referred to such cases under the name " appendicitis larvata," and Senator used the term " atypische appendicitis " for them. In America the rôle of the appendix in the production of gastric disorder has been recognised, not only in the work of the Drs. Mayo, but in the writings of Drs. Murphy, Ochsner, Deaver, and others. Three years ago I read a paper on this subject before the Royal Society of Medicine, and suggested that the term " appendicular gastralgia " is a convenient and descriptive one to apply to this condition.

Symptoms

Pain, varying in degree, is the prominent symptom in these cases. Some of the patients complain of what may be described as severe, continuous *discomfort* rather than pain, while some describe their *pain* as severe. In a large majority of the patients, the pain follows the ingestion of food, although the interval between the taking of a meal and the onset of pain is very variable. Some patients state that the pain comes on immediately after taking food, while in about one-third of the cases the interval between taking food and the onset of pain, varies from half an hour to three

hours. In most cases the pain is referred to the epigastrium, usually to the right of the middle line. Some of my patients stated that the pain radiated downwards to the right iliac fossa, while, with less definiteness, others said that the pain radiated downwards. In one interesting case the pain radiated to the left iliac region, and at the operation the cæcum and appendix were found in the left iliac fossa. This radiation of the epigastric pain to the lower part of the abdomen is a most important diagnostic symptom, and in my experience, is one which never occurs in gastric, or duodenal ulcer. Occasionally very definite " hunger pain " is a symptom. Sour eructations and vomiting are present in considerably more than half of the cases. Flatulence and a feeling of distension after food is a very constant feature. Hæmorrhage occurs as an occasional symptom (15 per cent. of my cases), and I think there can be little doubt that the hæmorrhage comes from the gastric mucous membrane. In one case, in which I opened the stomach, the whole of the mucosa was studded with numberless bleeding-points. The interior of the stomach was in the condition described as " weeping blood." Probably this condition of the mucosa is due to the irritation of hyper-acid gastric juice, or to the continual presence of gastric juice in the fasting stomach (hyper-secretion).

Periodical Exacerbations.—A noteworthy feature of these cases is, that in many instances (67 per cent.), there is a periodical exacerbation of the symptoms. As a rule the patients are not quite well during the interval between the attacks, but suffer from discomfort and flatulence after food.

Another striking feature is the long history in a large number of the cases. The average duration of illness in my cases is six years, although in one or two of them there was an interval of one or two years between the attacks. Tender-

ness in the epigastrium, usually to the right of the middle line, is an almost constant feature. In one-third of the cases there was tenderness over the appendix area as well as in the epigastrium. By far the larger majority of the patients are women.

Gastric Analysis.—The gastric findings are fairly constant. As a rule there is diminution or absence of free hydrochloric acid, but very occasionally there is hyperchlorhydria. The total acidity is usually above normal, the total chlorides are diminished, and the volatile acids slightly increased. Usually there is an excess of mucus, and sometimes the gastric contents are very thick and viscid. Hypersecretion is present in a considerable proportion of the cases.

The following is an illustrative case :

Female, aged thirty-seven years. Ten years ago the patient began to suffer from pain in the abdomen. She was said to have a " gastric ulcer." Ever since, she has suffered from pain coming on after food, with occasional free intervals. She used sometimes to make herself sick to relieve the pain. For the last three months she has been worse, as she always suffers from pain coming on an hour or less after food. She has had no vomiting, but has had sour eructations, and much flatulence. She is very constipated. In the morning the stomach contained 30 c.c. of yellowish fluid but no food residue. After test meal :

Total acidity	75
Total chlorides	0·318
Free HCl	0·010
Protein HCl	0·210.
Mineral chlorides	0·098
Volatile acids	6·8

At the operation the appendix was found to be bulbous, and bound down by dense adhesions. It contained a concretion. The mucosa was chronically inflamed, and in places hæmorrhagic. After operation her symptoms disappeared, and there was no hyper-secretion, the stomach being empty in the morning.

The Operation Findings.—As regards the condition of the appendix, as a rule, judged by its external appearance, it is not extensively diseased. The most common condition is that the appendix is bulbous, somewhat thicker than usual, and, on opening it, a narrowing or constriction is found near the base, the distal side of the constriction being slightly dilated and giving rise to the bulbous appearance. A concretion was found in exactly half my cases. In all of my cases, microscopic examination of the appendix showed the existence of chronic inflammation.

In some of the cases the disease of the appendix is more extensive than that described above. There may be minute hæmorrhages in the mucous membrane, or a definite ulcerated area of the mucosa. In one of my cases the appendix was enormously dilated, measuring $3\frac{1}{2}$ inches in circumference, the mucous membrane was ulcerated, and there were three large, very foul-smelling concretions in the lumen, as well as some blood-clot. This patient had had hæmatemesis as well as melæna.

There can be no doubt that an appendix, which on cursory examination, appears to present no abnormal features, may be the seat of disease fraught with grave danger to its possessor. On a number of occasions I have removed an appendix which, by an onlooker, might be regarded as " normal," and yet, subsequently, have been able to demonstrate to those present, the existence of disease within. Such an appendix usually presents the following features : It may not be obviously larger than usual, but inspection shows, that instead of being a uniform pale yellowish colour, its surface is covered with a branch-work of minute blood-vessels—evidence of chronic inflammation. Careful palpation may detect that the appendicular coats are thicker than usual. On cutting open the appendix, usually it is found to contain fæces, or a concretion, the mucosa is con-

gested, sometimes petechial, or even definitely ulcerated. Microscopic examination shows the existence of chronic inflammation. However innocent-looking externally, an appendix which contains a concretion, or the mucosa of which is so damaged as to permit of bacterial invasion, is one which, at any time, may lead to a sudden attack of appendicitis.

The evidence that the gastric symptoms are due to the appendicular disease is threefold. First, the majority of the patients are cured by appendicectomy. The pain and discomfort are relieved, and the vomiting ceases. Secondly, usually there is an improvement in the character and quality of the gastric contents, which indicates the close physiological connection between the stomach and the appendix. Thirdly, the frequency with which patients who come under observation with an acute attack of appendicitis, give, on cross-examination, a history of previous gastric trouble, is certainly significant. A number of these patients state, that for years they have suffered from what they describe as bilious attacks—that is, attacks of vomiting, with discomfort after food, in the intervals between the attacks. More frequently, the patients state that for a considerable time before the acute attack of appendicitis, they have had to be careful as to their diet, otherwise they have suffered from indigestion. Dr. Soltau Fenwick has pointed out that before he recognised the connection between appendicular disease and gastric symptoms, he was acquainted with a peculiar type of hyper-secretion in which, frequently death occurred from appendicitis. Some years ago I had an interesting case of this type. I operated on a lady who was supposed to have a gastric, or duodenal ulcer. On opening the abdomen I could find no lesion of the stomach or duodenum. The symptoms were clear and definite, and, as the patient had had hæmorrhage, I thought that an ulcer must exist undiscover-

able from external examination of the stomach. Accordingly I opened the stomach—a procedure of which, by the way, I now most strongly disapprove—and explored it from within. I found the whole mucous membrane in a state of extreme congestion, and studded all over with hundreds of minute bleeding-points. Accordingly I performed a gastro-jejunostomy, and the patient was relieved at once of her symptoms, remained perfectly well for about eighteen months, but then had an attack of acute appendicitis, and narrowly escaped with her life. At that time I did not recognise the association of appendicular disease with the causation of gastric symptoms, but on looking back I have little doubt that the original gastric symptoms were secondary to chronic disease of the appendix. In this particular case gastro-jejunostomy was a success in so far as it relieved the patient of her gastric symptoms; nevertheless, I am convinced that I made a mistake, in that I ought not to have performed gastro-jejunostomy, but to have removed the appendix. Some years ago I saw two cases of gastric catarrh in children, due to latent disease of the appendix, which I did not recognise. In one of these the gastric catarrh was attributed to adenoids, and the boy was sent to me by a well-known physician to have his adenoids removed. This was done, but the boy was not a great deal better, and five months later he had a very acute attack of appendicitis. After appendicectomy he rapidly improved, and since has remained well. The other case was that of a girl who, for several years had been subject to attacks of gastritis without obvious cause. Later, she had definite tenderness over the appendix, and appendicectomy was performed. Since the operation, eight years ago, she has had no further trouble.

Differential Diagnosis.—The chief conditions from which appendicular gastralgia has to be distinguished are duodenal ulcer, gastric ulcer, and gall-stones. Of these, **duodenal**

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ulcer is the disease which gives rise to the greatest difficulty in the differential diagnosis. The symptoms produced by duodenal ulcer, and by the cases of latent appendicitis to which I am referring, are very similar in many respects. There is often the same history of recurrent attacks ; both duodenal ulcer and appendicular disease may give rise to hyperchlorhydria, and also to entire absence of free HCl. Even " hunger pain " may be a symptom of appendicular disease. The points which aid us in distinguishing the two diseases appear to me to be : First, in duodenal ulcer the patient usually is free from symptoms between the attacks ; in appendicular gastralgia, even between the attacks, the patient suffers from flatulence and discomfort after food. Secondly, the radiation of the epigastric pain towards the lower part of the abdomen is highly suggestive, if not diagnostic, of appendicular trouble. Thirdly, the existence of tenderness over the appendix sometimes throws light on an otherwise doubtful case. Fourthly, in many cases of appendicular gastralgia the alteration of the gastric contents is not commensurate with the severity and duration of the symptoms. Let me explain what I mean by this. In a number of the cases which I have examined, the gastric contents have been little altered chemically. This is not often the case in duodenal ulcer. In my experience duodenal ulcer most commonly produces, or is associated with, hyperchlorhydria, at any rate in the early stages ; later, occasionally, there is absence of free HCl. Appendicular disease apparently may exist for a considerable time without a marked alteration of the amount of free HCl. The combination of marked symptoms of duodenal or gastric ulcer, with a negative gastric analysis, is suggestive of chronic appendicular disease. The cases of appendicular gastralgia associated with hyperchlorhydria present the greatest difficulty in diagnosis. My impression is, that hyperchlorhydria,

secondary to appendicular disease does not give rise to the severe pain which usually is associated with the hyperchlorhydria which accompanies duodenal ulcer. Fifthly, a point on which too much stress should not be laid, is, that duodenal ulcer is far more common in men; appendicular gastralgia appears to be more common in women.

In **gastric ulcer**, food, especially milk, often gives relief for the time being, the pain recurring one or two hours later. In appendicular gastralgia the onset of pain is more variable; food, even milk, usually produces pain or discomfort at once. In gastric ulcer, usually the motility of the stomach is not impaired, unless the ulcer be near the pylorus, in which case often there is food retention. In gastric and duodenal ulcer the pain is rarely so severe and continuous that the patient has to take to bed. In several of my cases of appendicular gastralgia, the patients have had to lie up on account of the continuous and exhausting character of the pain. Another point which is helpful in the diagnosis is this: the administration of bismuth and alkalis, in my experience, has little, or no influence on the pain and discomfort due to appendicular disease.

(For the points of difference between appendicular gastralgia and gall-stones, see p. 167.)

How are the Gastric Symptoms Produced?—When we attempt to explain why appendicular disease should produce gastric symptoms, we are entering into the region of speculation. I think I have brought forward evidence to show that there is a close connection between the appendix and the stomach, but as to the exact nature of this connection I am not prepared to dogmatise. Dr. W. J. Mayo thinks that appendicular disease causes a protective spasm of the pylorus. Possibly this is the explanation of those cases in which there is hyperchlorhydria. As Dr. Fenwick has pointed out, the symptoms are those of hyper-secretion. Exactly how the

hyper-secretion is caused, we are not in a position to say. All we know at present is, that in some way, the appendix does affect the gastric secretion. Personally, I think that intestinal stasis, rather than pyloric spasm, is the condition in these cases. The symptoms may be regarded as toxic, the result of the intestinal stasis. This view seems to be supported by the frequency with which the duodenum is found markedly dilated at operation, and by the fact that in a number of the cases the stomach also is dilated. Further, as a rule, the quantity of gastric contents evacuated after a test-meal is greater than in healthy individuals, the percentage of volatile acids is increased, and, in addition, flatulence and constipation are prominent symptoms.

The Indications for Operation.—That latent appendicular disease gives rise to gastric symptoms is now established definitely. The symptoms are those of hyper-secretion or the so-called " acid dyspepsia." I need hardly state that I do not suggest that all, or even a majority, of the cases of acid dyspepsia require operation, unless the symptoms are persistent or recurrent. Persistent acid dyspepsia, in my opinion, is secondary to some definite organic lesion. When we have reason to believe that gastric symptoms are due to appendicular disease, then usually operation is advisable, provided that medical treatment has had a thorough trial and has failed. I hold it as a cardinal principle, that in all cases of gastric disorder, except when there is evidence of some gross lesion, such as carcinoma, pyloric stenosis, or hour-glass stomach, it must be premised that medical treatment has been tried and failed. And when I speak of medical treatment I mean, not a few weeks of drug-taking, but a course of rest and careful dieting, extended over at least six months.

It is the cases in which there are no physical or chemical signs, which require careful consideration. In those cases in

which, in addition to the gastric symptoms, there is definite and persistent tenderness over the appendix, operation usually is advisable. I confess that I have an old-fashioned prejudice against operating when pain is the only symptom. At the same time, I recognise that there are such cases in which operation is not only justifiable but necessary. I take it that all are agreed that it is right to operate in cases of so-called " appendicular colic " when such attacks are recurrent. Surely it is equally justifiable to remove the appendix, when the patient suffers from recurrent attacks of gastralgia attributable to disease of the appendix, provided medical treatment has been tried and has failed. And in this connection I would point out again the diagnostic significance of radiation of epigastric pain to the lower part of the abdomen. I would insist, however, that the pain must be definite, and such that it interferes seriously with the patient's enjoyment of life.

CONCLUSIONS

(1) Appendicular disease may give rise to symptoms which mimic closely the supposed symptoms of gastric and duodenal ulcer.

(2) The prominent symptom is epigastric pain or severe discomfort after food ; in many cases there are sour eructations, vomiting, and even hæmatemesis and melæna.

(3) The radiation of epigastric pain to the lower abdomen is very suggestive of appendicular trouble.

(4) Gastric analysis reveals, in a few cases hyperchlorhydria, in others a normal amount of free HCl, but in the majority a marked diminution or absence of free HCl. As a rule there is an increase of the volatile acids, and, in many cases, evidence of hyper-secretion.

(5) Some cases of hyper-secretion or acid dyspepsia, and

many cases of supposed gastric or duodenal ulcer, are cases of latent appendicular disease.

(6) The evidence that the gastric symptoms are due to appendicular disease is threefold : (*a*) the majority of the patients are cured by appendicectomy ; (*b*) the influence which appendicectomy has on the gastric contents ; (*c*) the frequency of a history of previous gastric symptoms in those who have an attack of acute appendicitis.

(7) Probably the symptoms are the result of intestinal toxæmia due to intestinal stasis. In the early stages, the effect on gastric secretion possibly is due to pyloric spasm, but more probably to the controlling influence of the appendix on the effluent from the ileum.

(8) Appendicular gastralgia is apparently more common in women than in men.

(9) The important lessons to be learnt from these cases are : (i) That no operation should be performed on the stomach, except when a definite organic lesion of the stomach or duodenum exists. Gastro-jejunostomy will not cure appendicitis. (ii) That in all operations for supposed gastric or duodenal ulcer, the condition of the appendix should be investigated carefully.

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CHAPTER XXIII

SYPHILIS AND TUBERCLE OF THE STOMACH

Syphilis of the stomach is rare, although probably more common than is generally believed. It is more frequent in men than in women, and the patients usually are between the ages of thirty and forty-five years. It is manifested in three forms—gummata, chronic gastritis, and pyloric stenosis.

1. **Gummata.**—Gummata originate in the submucous tissue, and rarely attain a large size. As a gumma increases in size it undergoes caseation, the overlying mucosa becomes infiltrated, thinned, and finally destroyed, resulting in a gummatous ulcer. Such an ulcer is irregular in shape, with thickened, undermined edges, its base covered with a yellow slough. The surrounding mucous membrane is of a reddish slate-colour, owing to chronic inflammation. Ulcers may also result from interference with the vascular supply of circumscribed areas of the gastric mucosa, due to obliterative arteritis.

2. **Chronic Gastritis.**—As Dr. Soltau Fenwick points out, chronic gastritis may ensue either as a direct, or as an indirect result of syphilis. The *indirect* variety is the more common, and " is due either to embarrassment of the gastric circulation from disease of the liver or spleen, to lardaceous degeneration of the vessels of the stomach, to secondary disease of the kidneys, or to the specific cachexia. Chronic inflammation of the stomach *directly* dependent on the systemic infection, occasionally results from repeated attacks of an acute character during the early phase of the com-

plaint, but as a rule it appears only at an advanced stage of the disease, and is often associated with gummatous lesions of the bones, liver, or testes."

3. **Pyloric Stenosis.**—Pyloric stenosis may arise either from cicatrisation of ulcers in the neighbourhood of the pylorus, or from gummatous infiltration of the pylorus. It is a rare condition. I have seen only one example.

The patient, a man aged forty-three, was under the care of my colleague, Dr. Soltau Fenwick, suffering from pain coming on two hours after food, and vomiting. The vomiting gradually increased in frequency, until it occurred regularly once in the day and once during the night. The quantity vomited was often as much as two pints. He had a large, splashy stomach with visible peristalsis. On passing a tube in the early morning, four pints of fluid, containing much food remains, was recovered. Gastric analysis of a test-meal gave the following results:

Total acidity	88
Total chlorides	0·365
Free HCl	0·048
Protein HCl	0·204
Mineral chlorides	0·113

Dr. Fenwick diagnosed syphilitic disease of the stomach, and after a trial of anti-syphilitic remedies, advised operation. At the operation I found well-marked stenosis of the pylorus due to fibrous thickening. There was much fibrous thickening all round the liver, evidently the result of perihepatitis. I performed gastro-jejunostomy, and some years later, I heard that the patient was in perfect health.

Pyloric stenosis, the result of syphilitic disease in the vicinity of the pylorus, is a more common condition. I have met with two examples. In one case, that of a man aged thirty-three years, a gumma of the liver had become adherent to the first part of the duodenum, and caused obstruction. I drained the gumma and performed gastro-jejunostomy, which was followed by complete relief.

Symptoms.—The symptoms of a gastric ulcer due to syphilis are, pain after food and vomiting. Hæmatemesis is not common. As in all cases of visceral syphilis, anæmia is a prominent symptom, and often is very profound. The cachectic appearance of the patient is very striking. According to Dr. Soltau Fenwick these cases differ from the simple variety of ulcer in three particulars: the extreme severity of the pain and vomiting, the infrequency of hæmatemesis, and their resistance to ordinary treatment, and great tendency to relapse.

Treatment.—In cases of intractable gastritis, or gastric ulcer, the possibility of a syphilitic origin must be borne in mind, and if there be any suspicion of the presence of this disease, a Wasserman's test should be performed. If this be positive, a trial should be given to the administration of potassium iodide, and the intra-muscular injection of mercury, together with rest in bed and milk diet. If these prove ineffectual, or the symptoms recur, recourse should be had to gastro-jejunostomy. As has been pointed out by Dr. Fenwick, the possibility should be kept in view that the symptoms of gastric irritation in a syphilitic subject may be due, not to the specific malady, but to the prolonged administration of mercury, and potassium iodide.

In pyloric stenosis, medical treatment is of no avail; gastro-jejunostomy is indicated.

Tubercle of the Stomach

Primary Tubercle of the stomach is very rare. Usually it is secondary to, or associated with, tubercle of other organs.

The symptoms are indefinite, but may suggest carcinoma, to which the gastric findings bear a close resemblance.

As a rule, surgical treatment is of no avail, although two

cases have been recorded of successful resection of the pylorus for stenosis of tuberculous origin.

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CHAPTER XXIV

GASTRIC ATONY AND GASTROPTOSIS

IN gastric atony there is impaired contractile power of the muscular tone of the stomach, so that the stomach becomes dilated, and unable to expel its contents within the usual period after taking a meal.

Causes.—The most frequent causes are : (1) over-distension of the stomach with large meals, (2) too frequent meals, (3) over-indulgence in strong tea, and (4) the extra strain thrown on the stomach by a purely vegetarian diet. Malnutrition, insufficient exercise, a sedentary life in badly-ventilated rooms, and prolonged use of drugs such as iodide of potassium, quinine, and arsenic, are also predisposing causes.

Gastric atony may be secondary to such diseases as anæmia, disease of the heart and liver, and chronic gastritis. Last, but not least, gastric atony is an invariable accompaniment of chronic constipation.

Symptoms.—Discomfort, and sense of weight or fullness, coming on immediately after food, and lasting for two or more hours, is the prominent symptom. The discomfort is referred to the epigastrium, and is accompanied by distension, so that often it is necessary to loosen the clothes. Belching of gas is a constant and troublesome feature of the condition, often forming the subject of the patient's chief complaint. Fluids generally cause more discomfort than solids. Constipation is always present. Dr. Soltau Fenwick has pointed out the significance of the presence of a stomach

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splash as indicating the existence of impairment of the gastric motility. " In any case," he writes, " of myasthenia, whether primary or secondary, clapitage may be observed from the commencement of the meal until the expiration of three or more hours, and, in severe instances, it may be only in the early morning that manipulation of the epigastrium fails to produce the characteristic sound."

Another test for atony is as follows : if, on an empty stomach, the patient take a wine-glassful of water, and splashing can be elicited, we may conclude that gastric atony is present.

Gastric Analysis.—The amount recovered after a test-meal is increased : instead of amounting to 150 c.c. it may be as much as 250 c.c. or more. In the later stages of the disease, food may be found in the stomach in the early morning.

The gastric contents are sour-smelling, but the chlorides do not appreciably deviate from the limits of health. When, however, the trouble is of old standing, or chronic gastritis is present, free hydrochloric acid may be absent, and the protein hydrochloric acid may be diminished. The condition must be distinguished from that of pyloric stenosis. In the latter disease the patient is more comfortable on a liquid diet, while in gastric atony a carefully-regulated solid diet gives more relief. In pyloric stenosis vomiting is frequent and severe, and malnutrition of the patient is pronounced.

Treatment.—The diet must be regulated carefully. Sweet or fatty foods and the amount of fluid taken must be limited strictly. Unless there be gastric catarrh, lavage is not indicated, but massage often is very helpful. Drugs are not of much service, with the exception of dilute hydrochloric acid when there is deficiency of gastric juice. Gastro-jejunostomy is contra-indicated. It is of no use in gastric atony.

PLATE LII.
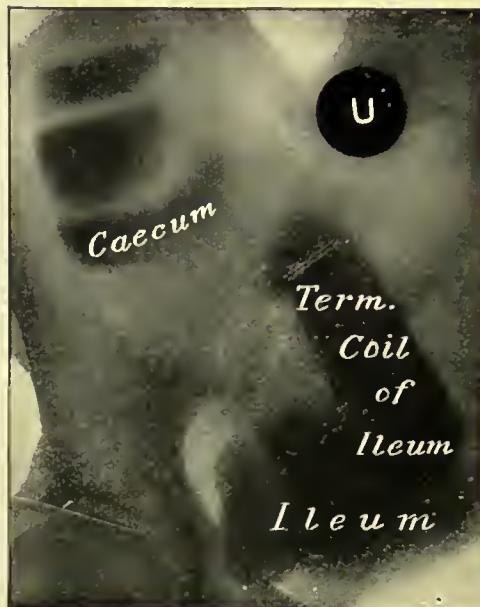
FIG. 81.—Taken on the couch seven hours after bismuth meal. The greater part of the bismuth was still in the stomach. The terminal coil of the ileum is shown greatly dilated; a small amount of bismuth has entered the cæcum and ascending colon. *U.*, umbilicus, marked by a penny. At the subsequent operation the appendix was found to be fixed at its base by bands in such a way that it obstructed the ileum at its junction with the cæcum. On removing the appendix the ileal obstruction was freed at once. —By Dr. A. C. JORDAN.

FIG. 82.—Ileal kink. Taken twenty-five hours after a bismuth meal, showing a long terminal coil of the ileum rising out of the pelvis; fixed by adhesions at the point *X*, close to the ileo-cæcal entrance. There was tenderness to pressure at this point. The dropped transverse colon is also shown. (Confirmed by operation.) —By Dr. A. C. JORDAN.

The most important symptom to be treated is constipation. The best drug is liquid paraffin, an ounce being given the last thing at night and a similar quantity in the early morning.

Although surgery rarely is indicated in the treatment of gastric atony, we must bear in mind that constipation may be the primary condition, and that constipation may be due to the presence of one of the kinks described by Sir Arbuthnot Lane. The existence of these kinks has been questioned by some authorities, but as to their presence and injurious effects, I have no doubt whatever. These kinks can be demonstrated by means of the X-rays. I know from experience of a number of cases, that the division of them, or their elimination, by means of a short circuit between the ileum and pelvic colon, is followed by most gratifying results.

GASTROPTOSIS

Gastroptosis, or downward displacement of the whole stomach, rarely exists except in association with dislocation of other abdominal organs. The condition is often termed Glénard's disease, after the physician who was the first to describe it.

The lengthening and stretching of the ligaments and mesentery of the stomach may be so great, that the lower curvature may reach almost to the pubes, while the upper curvature may be as low as the level of the umbilicus.

Gastroptosis must be distinguished from dilatation of the stomach. In dilatation, the distance between the upper and lower borders of the stomach is much greater than normal, while in gastroptosis this distance is increased little, if at all. Dilatation, with atony of the stomach, may be present in addition to the displacement.

The limits of the stomach are best determined by inflating the stomach with air, either by means of a stomach-tube, or by administering separately, the two halves of a seidlitz powder.

Two Varieties of Gastroptosis are described, the congenital and the acquired.

1. **Congenital Gastroptosis.**—Usually this is accompanied by a general enteroptosis. Spiller, who has devoted particular attention to the condition, speaks of " an universal asthenia of a congenital nature, or habitus enteroptoticus." Patients thus affected have a stooping carriage, protuberant, lax abdomens, flat chests, and dyspeptic symptoms. Usually they are females in the early adult period of life.

2. **Acquired Gastroptosis.**—This is due to mechanical causes, such as frequent pregnancies, with insufficient rest after parturition, tight-lacing and other improper modes of dress, and to malnutrition, caused by insufficient, or unsuitable food.

Symptoms.—Patients complain of a sense of weight in the abdomen, fullness and discomfort after meals, sometimes nausea, and occasionally vomiting. Constipation is always present, sometimes of a very obstinate type.

The gastric symptoms are due to impaired motility, or to disordered secretion, or to both. The muscular tone of the stomach is impaired, there is delay in the passage onwards of the gastric contents. The stomach performs its work efficiently as to final results, but inefficiently as to time : it empties itself slowly, but completely, so that there is never stagnation, as is the case with pyloric stenosis.

The secretion of the stomach is increased, so that as a rule, gastric juice is present in the fasting stomach, and usually there is some hyper-acidity.

Treatment.—The treatment of gastroptosis is not surgical. In itself it is not a disease, many patients having marked

gastroptosis, unaccompanied by any symptoms referable to the stomach. Symptoms, when present, are due to the want of tone of the muscular tissue of the abdominal wall and viscera, and to the general condition of the patient, and not to the displacement of the stomach.

Therefore I do not believe in performing gastropexy, or other operation for gastroptosis. I regard the various " pexies " as among the most unsatisfactory operations in surgery.

Moreover, the patients are generally of a type in which any surgical procedure should be avoided, if possible. They are only too ready to submit to operation, until, as Dr. W. J. Mayo puts it, " they have had all the movable organs fixed, and the removable ones removed." They rarely are benefited, and nothing results but failure and discredit.

The Treatment of Gastroptosis is twofold : first, general, and secondly, mechanical.

General.—Our aim should be to improve the general health of the patient. Abdominal massage is of service, and in some cases a rest cure is indicated. Hyper-secretion is treated by the administration of plenty of fat in the food, and perhaps the most important of all, constipation must be relieved. This is best effected by the administration, night and morning, of an ounce of pure liquid paraffin. Needless to say, the diet must be of the plainest description, starchy foods being rigorously limited.

Mechanical.—A well-fitting abdominal belt gives great comfort to the patient. It should fit firmly under the lower abdomen, and be kept in place, either by perineal straps, or by being made to fit round the upper part of the thighs. An aluminium plate on the lower part of the abdomen, moulded to fit accurately, adds to its efficacy. Such a belt gives relief, not by any pushing up of the displaced viscera, but by diminishing the amount of blood in the splanchnic area, and so raising the general blood-pressure.

APPENDIX

THE USE OF THE STOMACH-TUBE

THE stomach-tube is made of soft red rubber with two lateral, oval openings near its tip. The tip should be solid, so that material does not collect below the lateral openings. The edges of the openings should be rounded, so that they
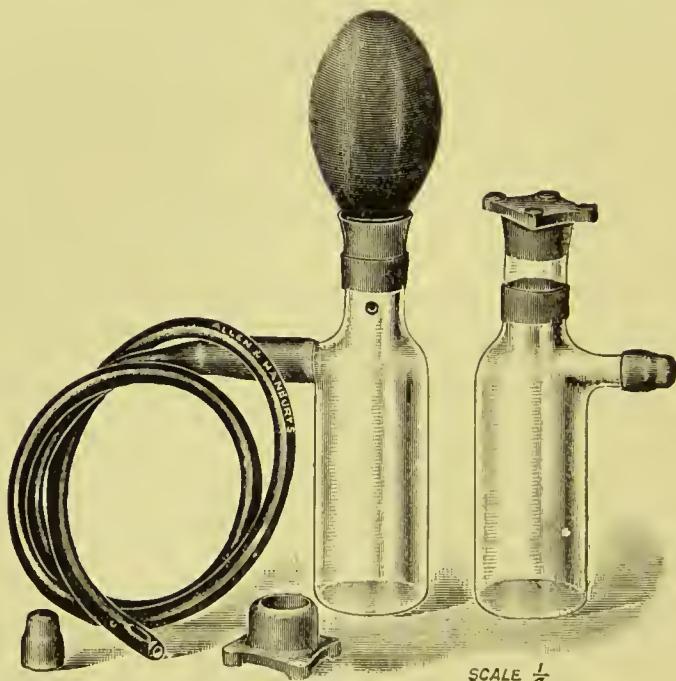
FIG. 83.—Senoran's stomach evacuator.

may not injure the gastric mucous membrane. As large a tube as possible should be used, as the larger the tube, the easier it is to pass, and the better it is tolerated. For adults a No. 32 or 36 (French scale) tube is a suitable size. Before use the tube is placed in hot water. No other lubricant is necessary. The patient is seated in a chair, with the head

inclined slightly forwards. Any artificial teeth are removed. The tube is inserted into the mouth, and as soon as it touches the posterior pharyngeal wall the patient is directed to swallow, and the tube is pushed rapidly onwards until it reaches the stomach. If the patient be instructed to breathe rapidly through the nose, his attention is directed away from the unpleasantness of the tube, and it causes less discomfort than would otherwise be the case. The best method of withdrawing the stomach contents is by means of a Senoran's aspirator (Fig. 83). This consists of a glass bottle with a capacity of 200 c.c., to the mouth of which is fitted a compressible rubber bulb. From the neck of the bottle projects a nozzle, to which the stomach-tube can be attached. On the opposite side of the neck is a small hole. If the bulb be compressed, and the hole in the neck covered by the finger tip, a partial vacuum is produced in the bottle, and the stomach contents are sucked out.

THE METHOD OF PERFORMING GASTRIC LAVAGE

This is best carried out with Senoran's aspirator. The stomach-tube is passed and attached to the bottle. The bottle is filled with warm water. The finger is then pressed on the hole in the neck and the bulb is compressed. This drives the water from the bottle into the stomach. Then, by using the bottle as an aspirator, the fluid can be withdrawn from the stomach into the bottle. The bottle is emptied and refilled with warm water, which in turn is run into the stomach. The process is repeated until the water returned is clear.

THE METHOD OF INVESTIGATING THE MOTOR FUNCTIONS OF THE STOMACH

One method of investigating the motor functions is given in Chapter I. (p. 6). Another simple method, but less instruc-

tive, is that of Ewald. The procedure is as follows : Fifteen grains of salol are given with the food, and the urine tested for salicylic acid at intervals. Salol is split up by the alkaline pancreatic juice, but it is not acted on by the gastric juices. Therefore, salicylic acid cannot appear in the urine until the salol has reached the duodenum. Normally, after the administration of salol, salicylic acid appears in the urine after an interval varying from forty to seventy minutes ; in cases of motor insufficiency the acid will not be found until after a longer interval.

The test for salicyluric acid is made by putting a drop of urine on a filter paper, and then applying a drop of ferric chloride (10 per cent. solution) to the moist spot. If salicyluric acid be present a violet coloration will appear at the edge of the moist spot.

QUALITATIVE EXAMINATION OF GASTRIC CONTENTS

Reaction.—The reaction of the filtered gastric contents is tested by means of litmus paper. Normally the reaction should be acid, but it may be alkaline or amphoteric. If the reaction be acid, the filtered contents are tested for the presence of free hydrochloric acid. For this purpose one of several reagents may be used.

Congo Red.—A solution is prepared by dissolving one gramme of congo in 100 c.c. of distilled water. This solution turns blue on the addition of free hydrochloric acid. It is said that free hydrochloric acid, present to the amount only of 0·0009 per cent., is detected by this reagent.

It is more convenient to use red congo paper. This is made by saturating filter paper with an alkaline solution of congo, and allowing the paper to dry. Then it may be kept for use as required. The paper turns blue on the addition of filtered gastric contents containing free hydrochloric acid.

It is a less delicate test than the solution, as the blue coloration does not appear unless 0·01 per cent. of free hydrochloric acid be present in the gastric contents. Inasmuch as free lactic acid or acetic acid turn congo paper blue, occasionally the reaction for free hydrochloric acid is present, although by quantitative analysis, free hydrochloric acid is proved to be absent.

Dimethyl-amido-azo-benzol Test for Free HCl.—This test consists of a 0·5 per cent. alcoholic solution of dimethyl-amido-azo-benzol. A few drops of the reagent are placed in a test-tube or porcelain dish, and a few cubic centimetres of filtered gastric contents are added. If free hydrochloric acid be present the solution turns a cherry red. Organic acids, if present in a greater percentage than 0·5 per cent., will give the cherry colour.

Gunzberg's Test for Free HCl.—Gunzberg's solution is made up as follows :

Phloroglucin	.	.	.	.	2 grammes
Vanillini	.	.	.	.	1 ,,
Alcohol absol.	.	.	.	.	30 ,,

This solution must be freshly prepared. If, however, the two reagents be made up separately, they may be kept for some months, and mixed together for use when required. To make the test, three drops of filtered gastric contents are placed in a porcelain capsule, and a few drops of the reagent added. The capsule then is heated very gently over a spirit flame. If free hydrochloric acid be present, as the fluid evaporates a beautiful cherry red appears round the edges of the mixture. This test is a very delicate and reliable one. Solutions of free hydrochloric acid as weak as 0·01 per cent. give the reaction. No organic acids give the reaction.

Test for Lactic Acid.—The presence of lactic acid is detected by Uffelmann's reagent. Ten cubic centimetres, of a one in twenty solution of carbolic acid, are placed in a

test-tube, and a few drops of liquor ferri perchloridi added ; this mixture is a deep amethyst blue. Enough distilled water is added to render the fluid transparent, and 5 c.c. of the resulting solution are placed in a test-tube. To this a few drops of filtered gastric contents are added. If lactic acid be present, a canary yellow or greenish-yellow colour results.

It is more delicate to make the test for lactic acid, after extracting the gastric contents, with ether. The ether dissolves the lactic acid, other substances being left behind. The ethereal solution is evaporated on a water bath, and the residue extracted with water and tested as described above. In the presence of lactic acid the solution turns green.

Strauss' Test.—A more delicate test is that of Strauss. If made with a special Strauss funnel the test is a very simple one. The funnel has a stop-cock at its lower end. Above the stop-cock two marks are engraved on the glass, one mark 5 c.c. and the other mark 25 c.c. above the stop-cock. At the upper end of the funnel is a glass stopper. To apply the test, filtered gastric contents are run into the funnel up to the 5 c.c. mark, and ether added up to the 25 c.c. mark. The glass stopper is replaced and the funnel is shaken vigorously. It is then allowed to stand until the ether has separated from the gastric contents. The stop-cock then is opened and the fluid allowed to run out slowly until the remaining fluid reaches to the 5 c.c. mark. The funnel is now filled up to the 25 c.c. mark with distilled water, and a few drops of tincture of iron chloride added. If lactic acid be present, the mixture, on being shaken, turns a deep or pale green, according to the amount of acid present.

Gmelin's Test for Bile Pigment.—Ten cubic centimetres of fuming nitric acid are placed in a test-tube. By means of a pipette an equal quantity of filtered gastric contents is floated on the nitric acid. If bile pigment be present, a

green ring is formed at the junction of the nitric acid with the gastric contents.

The colour becomes more marked, if the test-tube be put aside for five or ten minutes.

Carmine Red Fibrin Test for Pepsin.—This method is described by Harley and Goodbody as follows :

" Some fibrin from bullock's blood is quickly washed free from all traces of colouring-matter, this being important so as to avoid decomposition of the fibrin. The fibrin is then dried with alcohol and ether, and stained with an alcoholic solution of carmine-red until the colour does not deepen. Any excess of carmine is removed by washing in water, and the fibrin thus prepared is kept in glycerine.

Method of carrying out the Test.—Three test-tubes are prepared as follows :

(1) Four c.c. of filtered gastric contents.

(2) Two c.c. of filtered gastric contents and two c.c. of a four per mille hydrochloric acid solution.

(3) Two c.c. of filtered gastric contents and two c.c. of distilled water.

A small piece of carmine fibrin, which has been washed clear of glycerine, is placed in each of the test-tubes, which are then placed in a water bath at 37° C., being taken out and shaken every two or three minutes. The time when the fluid is first coloured by carmine is noted. Pepsin, in the presence of hydrochloric acid, dissolves the fibrin, converting it into soluble fibrin, thus liberating the carmine which becomes diffused through the fluid, and indicates that digestion has commenced.

If pepsin and hydrochloric acid are present in the gastric contents in normal amounts, it will be found that test-tube No. 1 will show a red coloration in from three to five minutes. In the event of the hydrochloric acid in the gastric contents being normal, the additional quantity of hydro-

chloric acid in test-tube No. 2 will tend to make it excessive, and thus hinder digestion.

In hypochlorhydria, or in cases where the proenzyme and not pepsin is present, test-tube No. 2 will show a red coloration before the other test-tubes.

In hyperchlorhydria, it will be found that test-tube No. 3, which has been diluted with water, will show distinct digestion of the fibrin before test-tubes Nos. 1 and 2, since the pepsin will not act as rapidly if hydrochloric acid is in excess in the gastric contents.

As examples of these three conditions, we will give three typical cases.

(A) Euchlorhydria

(1) Red coloration in 3 to 5 minutes.
(2) Red coloration in 3 to 5 minutes.
(3) Red coloration in 5 to 10 minutes.

(B) Hyperchlorhydria

(1) Red coloration in 7 to 15 minutes.
(2) Red coloration in 3 to 7 minutes.
(3) Red coloration in 3 to 12 minutes.

(C) Hypochlorhydria

(1) Red coloration in 10 to 30 minutes.
(2) Red coloration in 5 to 30 minutes.
(3) Red coloration in 30 to 120 minutes.

QUANTITATIVE EXAMINATION OF GASTRIC CONTENTS

Total Acidity.—The usual method of estimating the total acidity of the gastric contents is that introduced by Ewald (*Klinik der Verdauungs Krankheiten*, 1893, ii. p. 34).

Method.—Ten cubic centimetres of gastric contents are measured carefully into an Erlenmeyer's flask or small

beaker. An equal quantity of distilled water is added, as well as a few drops of a one per cent. alcoholic solution of phenophthalein, to act as an indicator. From a burette a decinormal solution of sodium hydroxide is run in drop by drop, until the red colour, which the liquid in the flask gradually assumes, does not deepen on the addition of another drop. The number of cubic centimetres of sodium hydroxide used represents the total acidity in 10 c.c. of the filtered gastric contents. This figure multiplied by ten gives a number which expresses what is known as the total acidity. This number represents the acidity due to hydrochloric acid, plus organic acids, plus acid phosphates, in 100 c.c. of filtered gastric contents. In health it varies from fifty-five to sixty-five.

Estimation of Volatile Acids.—The simplest and best method is that devised by Professor Vaughan Harley (*Brit. Med. Jour.*, 1899, ii. p. 1271). The method is as follows:

" A certain quantity (preferably 25 c.c.) of unfiltered gastric contents is poured into a distillation flask, and 100 c.c. or more of water added. A current of superheated steam is driven through the flask, and a small flame placed underneath it. The superheated steam, raising the contents to boiling-point, drives off the volatile acids, which distil over a condenser into an Erlenmeyer flask containing about 10 c.c. of decinormal solution of sodium hydrate. After about thirty minutes, the Erlenmeyer flask is removed, and a few drops of neutral solution of litmus added to the sodium hydrate solution. The fluid is then titrated by rapidly running into it a decinormal solution of sulphuric acid, until a neutral reaction is obtained. The quantity of decinormal solution of sodium hydrate neutralised by the volatile acids is then easily obtained. Multiplying the number by four, if 25 c.c. of gastric contents were taken, will give the quantity of volatile acids in 100 c.c. of gastric contents."

QUANTITATIVE ESTIMATION OF HYDRO-CHLORIC ACID

Prout-Wynter Method.—This method is very accurate, but requires considerable practice to obtain reliable results. It is carried out as follows :

1. Place 10 c.c. of unfiltered gastric contents in a platinum capsule, and add 10 c.c. of a saturated solution of sodium carbonate ; heat to dryness on a sand bath, taking care that the contents of the capsule do not spurt ; then heat to redness, and, with distilled water, wash the contents of the capsule into a glass beaker, which should have a spout. Add sufficient nitric acid (about 10 c.c.) to neutralise the excess of sodium carbonate. Add 15 c.c. of silver nitrate (decinormal solution) from a burette, then filter into a larger beaker, being careful to wash the precipitate well with distilled water, at least three times. To the filtrate add a few drops of a saturated solution of iron alum, in distilled water, to act as an indicator. Next, slowly run in, from a burette, a decinormal solution of potassium sulpho-cyanide until the contents of the beaker have a faint, pinkish-yellow tinge. Subtract the number of c.c. of potassium sulpho-cyanide used from 15 c.c. (the amount of silver nitrate used), and this will give the number of c.c. of silver nitrate required to combine with the total chlorides in 10 c.c. of gastric contents. Multiply this number by 0·0365, and the result will be the amount of total chlorides in 100 c.c. of gastric contents.

2. Place 10 c.c. of unfiltered gastric contents in a platinum capsule, evaporate to dryness so as to drive off the free hydrochloric acid, being careful that the contents of the capsule do not become charred. Add 10 c.c. of a saturated solution of sodium carbonate and proceed in exactly the same manner as in No. 1.

3. Evaporate to dryness (in a platinum capsule) 10 c.c. of unfiltered gastric contents, heat to redness, and then proceed in exactly the same manner as in Nos. 1 and 2, *except* that only 10 c.c. of silver nitrate need be added, consequently the number of c.c. of potassium sulpho-cyanide used must be subtracted from ten instead of from fifteen.

1. The result of No. 1 gives the amount of total chlorides in 100 c.c. of unfiltered gastric contents.

2. The result of No. 2 gives the amount of all chlorides, except the free hydrochloric acid, in 100 c.c. of unfiltered gastric contents.

3. The result of No. 3 gives the amount of mineral chlorides only, in 100 c.c. of unfiltered gastric contents. Therefore :

> No. 1 = total chlorides.
> No. 1 – No. 2 = free hydrochloric acid.
> No. 3 – No. 2 = protein hydrochloric acid.
> No. 3 = mineral chlorides.

Töpfer's Method.—This method is much simpler than the Prout-Wynter method, but it is not nearly so accurate. Personally, I do not consider that it is of much value.

Method.—1. 10 c.c. of filtered gastric contents are placed in an Erlenmeyer's flask, and a few drops of a 1·0 per cent. alcoholic solution of phenopthalein added. The contents of the flask then are titrated with a decinormal solution of sodium hydroxide in the way described above for the estimation of the total acidity. The number of c.c. of sodium hydroxide used is multiplied by ten. Let **A** represent this figure.

2. Another 10 c.c. of filtered gastric contents are placed in a flask, and a few drops of a watery solution of alizarin added. On the addition of alizarin the contents of the flask turn yellow. The contents then are titrated with a decinormal solution of sodium hydroxide until the yellow

colour changes to violet. The number of c.c. of sodium hydroxide used is multiplied by 10. Let **B** represent this figure.

Then, **A** − **B** represents the amount of protein hydrochloric acid.

3. Another 10 c.c. of filtered gastric contents are measured into a flask and a few drops of dimethyl-amido-azo-benzol solution (0·5 per cent. in alcohol) are added. The mixture is titrated as before. The number of c.c. of sodium hydroxide used is read off and multiplied by 10. This gives the acidity due to free hydrochloric acid. Let **C** represent this figure.

As I pointed out in the description of the method of estimating the total acidity, the figure obtained represents the acidity due to hydrochloric acid, organic acids, and acid phosphates. Therefore :

A represents the total acidity due to hydrochloric acid, organic acids, and acid phosphates.

C represents the acidity due to free hydrochloric acid.

A − **B** represents the acidity due to protein hydrochloric acid.

A − (**B** + **C**) represents the acidity due to organic acids and acid phosphates.

QUANTITATIVE ESTIMATION OF PEPSIN

Mett's Method (Pawlow, *The Work of the Digestive Glands*, translated by W. H. Thomson, 1902, p. 26). Some fresh egg albumen is drawn into a fine capillary glass tube. The tube is placed in boiling water for five minutes. This coagulates the albumen. The tube and its contents are then cut into lengths measuring five centimetres each. To estimate the pepsin in the gastric contents, proceed as follows :

Some gastric contents (unfiltered) are placed in a test-tube or small beaker, and a piece of the prepared tube placed therein. The beaker then is placed in an incubator at a temperature of 37° C. for ten hours. At the end of

this time, if the capillary tube be examined, it will be seen that some of the albumen has disappeared from each end of the tube. The empty portions of the tube are measured by means of a millimetre scale and a magnifying glass. The square of the length of the column of albumen digested is the measure of the amount of pepsin in the gastric contents. For example, if the empty portion of the capillary tube measure two millimetres in length, the amount of pepsin present is represented by 2×2, that is, four parts of pepsin.

The unit is the quantity of pepsin which will digest one millimetre of egg albumen in a Mett's tube in ten hours, the tubes being immersed in 0·18 per cent. of free hydrochloric acid.

TEST FOR OCCULT BLOOD IN THE FÆCES

A piece of fæces the size of a hazel nut is mixed with water in a test-tube, so as to form a thick emulsion ; to this one-third of its volume of glacial acetic acid is added and the mixture shaken. Then 5 c.c. of ether are added and the two fluids are mixed by being shaken. On standing, the ethereal extract separates, and of this 2 c.c. are mixed with 2 c.c. of a saturated solution of benzidin (para-diamido-di-phenyl) in rectified spirit, and 2 c.c. of a solution of hydrogen peroxide. If blood be present in considerable quantity, the solution assumes immediately a deep-blue colour ; in lesser quantities the colour is less intense and green.

For three days before the test is applied the patient's diet must be free from blood-containing foods.

THE SERO-DIAGNOSIS OF CANCER

Draw 15 grammes of blood. It must not be more than thirty-six hours old or forty-eight at most. To 0·1 c.c. of the serum in a test-tube, 1 c.c. is added of a 5 per cent. suspension of hen's blood corpuscles, in a 0·85 per cent.

solution of sodium chloride. The test-tube is well shaken and placed in a water bath, and this in an incubator at a temperature of 37° C. A positive reaction consists in the entire absence of hæmolysis in the test-tube.

Kelling (*Archiv für Verdauungs Krankheiten*, Berlin, xvi. ii. No. 3; also *Jour. Amer. Med. Assoc.*, 1906, xlvii. p. 1963; 1910, iv. p. 1602) attaches great value to the sero-diagnosis of early cancer. He states that from an experience of the test in 1500 patients, 400 of whom had cancer, a positive reaction was never obtained in a healthy person. He maintains that a negative reaction excludes cancer. Rosenberg (*Deutsche Mediz. Woch.*, Berlin, June 27, vol. xxxvii. No. 26) finds that a positive reaction is found in many conditions other than cancer, and he is of opinion that the test is of little practical differential value.

THE SKIN REACTION IN CARCINOMA

Elsberg and Geist (*Amer. Jour. of the Med. Sciences*, Feb. 1910) have suggested a new method for detecting hæmolysis in patients suffering from cancer. Their method is to inject, subcutaneously, into the arm of the patient suspected to have cancer, five minims of a suspension of 20 per cent. washed human blood corpuscles in saline solution.

The corpuscles are attacked by the hæmolysins in the blood serum of the cancer patient, and a reaction appears in from two to eight hours. The reaction is the appearance, at the site of the injection, of a brownish-red or bluish colour.

It is said that the reaction occurs in nearly 90 per cent. of cancer cases.

THE LENHARTZ DIET IN THE TREATMENT OF GASTRIC ULCER

Professor Lenhartz keeps his patients at absolute rest, and even after hæmorrhage commences to feed them at once

on a diet of raw eggs and milk. On the first day he gives 100 c.c. (3½ ounces) of iced milk, in which is beaten up one raw egg. The mixture is sipped slowly out of a teaspoon, so that the patient takes a continuous supply of protein to combine with the hydrochloric acid of the gastric juice, and thus diminish any hyper-acidity which may be present. The quantity taken is increased day by day until it amounts to 800 c.c. (28½ ounces) of milk and six or eight eggs a day.

It is maintained that this diet contains a minimum amount of food with a maximum calorific value, so that by its use the patient's general nutrition is improved, and the healing of the ulcer promoted. Further, the continual presence, in the stomach, of protein food " fixes " the free hydrochloric acid, and thus diminishes the hyper-acidity which retards healing of the ulcer.

The diet is an excellent one, but personally, I do not approve of commencing it at once after gastric hæmorrhage. On several occasions I have seen an alarming hæmorrhage occur in a patient put on this regimen immediately after hæmatemesis. After hæmorrhage, therefore, I prefer to withhold all food by the mouth, and to give saline by the rectum, for three days. If, at the end of this period, the hæmorrhage have ceased, the Lenhartz diet may be commenced with safety.

SCHMIDT'S TEST DIET

In the morning, milk 500 c.c. (17½ ounces), or if milk does not agree, 500 c.c. of cocoa made from cocoa powder, 20 grammes (⅔ of an ounce) ; sugar 10 grammes (⅓ of an ounce) ; water 400 grammes (14 ounces) ; and milk 100 grammes (3½ ounces). In addition to the milk or cocoa 50 grammes of zweiback (six biscuits may be substituted for the zweiback).

In the forenoon, 500 c.c. (17½ ounces) of oatmeal gruel (made from oatmeal 1½ ounces, butter ⅓ of an ounce, milk 200 grammes (7 ounces), water 300 grammes (10½ ounces), and one egg).

At noon, 125 grammes (4⅓ ounces) raw weight of chopped beef broiled rare with 20 grammes (⅔ of an ounce) of butter, so that the interior still remains raw. In addition to the beef, 250 grammes of potato purée made from mashed potatoes 190 grammes (6⅔ ounces), 100 grammes of milk (3½ ounces), and 10 grammes (⅓ of an ounce) of butter.

In the afternoon, as in the morning.

In the evening, as in the forenoon.

This diet contains 102 grammes of albumin, 111 grammes of fat, 191 grammes of carbo-hydrates, a total of 2234 calories.

According to Schmidt, the chief inferences which may be drawn from examination of the fæces of a patient on this diet are the following :

1. The appearance of mucus in the fæces indicates the existence of an inflammatory condition of the mucous membrane.

2. If connective tissue appear in the fæces it is a sign of disturbance of the stomach digestion.

3. If macroscopic muscle remains appear in the fæces, this is a sign of a disturbance of intestinal digestion.

4. Remains both of connective tissue and of muscle, indicate disturbances in which stomach and small intestine participate.

5. Complete absence of the red and green colouring of fæces, when tested with mercuric chloride, occurs in fatty stools when the bile is completely shut off from the intestine.

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